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ARCHIVES

OF

OPHTHALMOLOGY

FOUNDED IN 1869 BY

DR. HERMAN KNAPP

EDITED IN ENGLISH AND GERMAN

BY

DR. ARNOLD KNAPP
OF NEW YORK

AND

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OF MUNICH

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DR. W. A. HOLDEN
OF NEW YORK

SEPTEMBER

NEW YORK
G. P. PUTNAM'S SONS, 2, 4, & 6 WEST 45TH STREET
AND NEW ROCHELLE, N. Y.
LONDON: 24 BEDFORD STREET, STRAND
WIESBADEN: J. F. BERGMANN'S Verlag
PARIS: J. B. BAILLIÈRE, 19 Rue Hautefeuille
1917

Entered at the Post-Office, New Rochelle, N. Y., as Second-Class Mail Matter

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ARCHIVES OF OPHTHALMOLOGY.

ON THE BINOCULAR METAMORPHOPSIA PRODUCED BY OPTICAL MEANS.

BY DR. J. A. LIPPINCOTT, MONTE CARLO (FORMERLY PITTSBURGH, PA.).

(With twelve figures in the text.)

IN a paper[1] published in these ARCHIVES many years ago I described the changes in the apparent forms of objects produced in binocular vision by means of spherical and cylindrical glasses. The subject was not entirely new because Wadsworth[2] had, in 1875, reported a case in which, after the fitting of cylindrical glasses, rectangular objects assumed a rhomboidal form, and Culbertson[3] had, in 1888, described similar phenomena under the title of "Binocular Astigmatism." The distortions of form were supposed to be exceptional and to be caused solely by cylindrical lenses. My paper demonstrated that, instead of being rare, binocular metamorphopsia may be observed in every case where there is binocular vision and that spherical as well as cylindrical glasses are capable of producing it.

My reasons for returning to the subject now are the following. In the first place no satisfactory explanation of the phenomena described in my paper has, in my opinion, been given. Dr. John Green,[4] it is true, made the pregnant

[1] "On the Binocular Metamorphopsia Produced by Correcting Glasses," ARCH OF OPHTHAL., March, 1889.

[2] *Trans. Amer. Oph. Soc.*, 1879, p. 342.

[3] *Journal of the Amer. Med. Assoc'n*, November 3, 1888.

[4] *Trans. Amer. Oph. Soc.*, 1889, p. 449.

suggestion that kindred appearances are stereoscopic in character, but he did not allude specifically to the facts presented in my article, and moreover, some of those facts are not at the first glance easily accounted for upon any theory of stereoscopic vision. Secondly I did not in my previous paper take account of the curious changes in the appearance of flat surfaces produced by prisms and I have seen no explanation of these changes that seemed to me at once clear and comprehensive. Furthermore, there is another form of binocular metamorphopsia induced by prisms, and not hitherto described, which is perhaps worthy of examination. Thirdly the facts of binocular metamorphopsia have, I think, a bearing on some disputed questions of retinal physiology relating especially to the projection of images and to the doctrine of corresponding points.

The terms "uniocular" and "binocular" are used in the present paper in a restricted sense, the former to express individuality, and the latter association, in function. The former presupposes only that kind of vision which is possible with a single eye but does not exclude the possibility of vision on both sides. The latter presupposes not only two seeing eyes but two eyes seeing in unison. Binocular metamorphopsia results from the uniocular metamorphopsia produced by spherical, cylindrical, or prismatic glasses, and its occurrence demonstrates the presence of binocular vision.[1] Donders's[2] description of binocular vision as resembling a chemical composition in contradistinction to a mechanical mixture on the ground that the resulting image differs from each of its constituent elements, although, as I believe, essentially *apropos*, must not be pushed too far. The characterizing feature of binocular vision is simply the comparing of the uniocular images without regard to the results of this comparison. In looking at a flat-surfaced object at a little distance the uniocular images are practically, if not theoretically, alike, and the binocular image differs from either of them only in being more distinct. The series of phenomena involved in the production of binocular metamorphopsia may

[1] This fact is the basis of the simple but effective procedure described in "New Tests for Binocular Vision," *Trans. Amer. Oph. Soc.*, 1890.

[2] Quoted by Parinaud, *La Vision*, 1898, p. 151.

be compared to what takes place in a judicial trial in which the testimony of the only two witnesses is conflicting in regard to one or more of certain essential points, and the judge, lending an ear to each of the witnesses, but trusting neither completely, reaches an independent and erroneous conclusion.

An analysis of the various forms of binocular metamorphopsia will be facilitated by a brief reference to some of the principles involved in their causation, particularly those relating to the estimation of *distance* and *size*.

Distance. Of all the methods of estimating distance the most important, especially for objects within a limited range, is that furnished by the degree of convergence of the visual axes. We converge more in looking at near than at distant objects. Hence we associate greater nearness with increased convergence. In the second place our estimate of the distance of an object is influenced by the size of the image. Convex spherical and cylindrical lenses, which enlarge the image, make the object appear nearer, while concave lenses have the opposite effect. Cylinders with axes horizontal seem to have more influence on the uniocular estimate of distance than when the axes are vertical.[1] *Size.* Our estimate of the size of an object is governed by our idea of its distance. If the object is supposed to be more distant than it really is it appears magnified. Experiences like the following have occurred to many. I was on one occasion reading, seated with my right side to a window which looked on a small court. To my great surprise I suddenly saw a bird of unusual shape and the size of a large hen flying along the opposite wall of the court. Almost in the same instant a fly, which was the cause of the phenomenon, appeared in front of my face. On the other hand, if the object is supposed to be nearer than it actually is it appears smaller. For example, if we converge, either by means of adducting prisms, or without them, for a point nearer than the object, and thus cause it to appear nearer than it really is, it seems smaller. This may account for the fact that some observers have thought that adducting

[1] In the natural objects by means of which we are accustomed to measure distance—mountains, trees, other men, etc.—the vertical dimensions predominate. Besides it is probable that the vertically acting muscles furnish a more delicate test than those which act horizontally.

prisms make objects appear more remote instead of nearer. As I shall have to refer rather frequently to the immediately foregoing observations it will be convenient to summarize them in the following rules:

Rule 1. The greater the convergence required to fix an object the nearer, and the less the convergence the more remote, the object appears.

Rule 2. The effect of convex lenses spherical or cylindrical is to make objects appear nearer, and the effect of concave glasses is to make them appear more remote.

Rule 3. When an object seems more distant than it really is it appears larger, and when it seems to be nearer than it is it appears smaller.

In studying the phenomena of binocular metamorphopsia we are compelled to take some account of disputed problems in regard to the nature of visual projection and of binocular vision. I shall, however, not at once enter upon a discussion of these questions but shall content myself for the moment with assuming 1st (with Parinaud)[1] that the images united in binocular vision are not the retinal but the projected images, and 2d that convergence, direct or indirect, is the essential factor in binocular fusion. Upon these two assumptions is based the following analysis of the facts grouped in accordance with their producing causes.

Group 1. *Effects of cylindrical glasses with axes vertical.* In looking at a square card through cylinders of unequal strength and with axes vertical, the binocular image is a trapezoid with the upper and lower borders non-parallel and the side corresponding to the eye furnished with the more convex or less concave glass higher than the other.

Let us examine one of the simplest examples, *e.g.* the right eye armed with a convex cylinder and the left with a plano. The image projected by the left eye, represented by A B C D (Fig. 1), shows the actual form of the square, while the image projected by the right eye, represented by A E F D, is widened in proportion to the strength of the cylinder. In the figure the images are superposed in such a manner that the left sides of the images coincide in the line A D, *i.e.* the visual axes

[1] *Annales d'oculistique*, 1904, Avril, Mai et Juin.

converge so as to meet in any point on this line. In this position of the eyes it is evident that they are in a state of greater divergence (less convergence) for the right sides of the

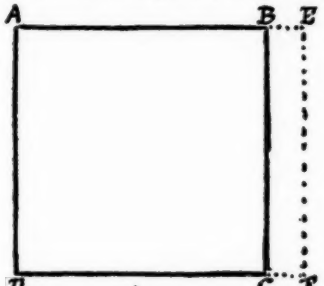

Fig. 1

images than for the left sides because, while both eyes are looking at A D, the left eye looks at B C and the right at E F. According to rule 1 the right side of the fused image appears more distant than the left, and consequently, by rule 3, higher.

Let us now suppose the images to be superposed in such a way that the right sides coincide, and let us replace the lines

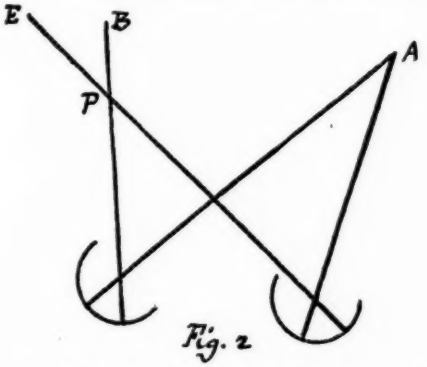

Fig. 2

representing the sides by single points on those lines. *E.g.* let the right sides (on which we converge) be represented by the point A (Fig. 2) and the left sides, which of course do not coincide, by the points E and B. The points A and E represent the image seen by the right eye, while the points

A and B represent that seen by the left. It is clear that the convergence is greater for the left sides than for the right, the lines of vision for the left sides crossing at the point P. Hence (rule 1) the left side of the fused image is seen nearer and (rule 3) lower than the right. If, instead of converging directly for either side, we fix the center of the square—*i.e.* superpose the images so that the centers coincide, the right side of the fused image will appear more distant, and the left side nearer, than the center, with the same effect on the form of the binocular image. The conditions are essentially the same for all combinations involving the use of cylinders with axes vertical. In each of them the fused image is higher on the side corresponding to the more convex or less concave glass.

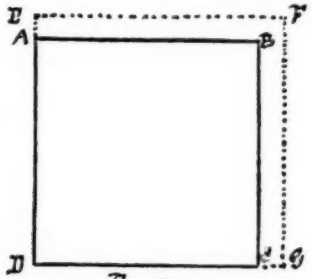

Fig. 3

Group 2. *Effects of spherical glasses.* The fused image appears higher on the side corresponding to the eye furnished with the more convex or less concave lens. Fig. 3 illustrates the effects of a convex lens on the right side and nothing on the left. E F G D represents the image projected by the right eye while A B C D stands for that of the left. The principles are the same as in Group 1. The convergence for the right sides of the images is less than for the left sides and therefore (rule 1) the right side of the fused image seems more distant, and hence (rule 3) higher, than the left; and this holds good no matter what part of the square is fixed.

But, curiously, a spherical glass produces less distortion of the square than a cylinder, axis vertical, of the same strength. If the metamorphopsia is due to the difference in the convergence for the two sides the distortion ought apparently to be

the same, because, it might be supposed, the widening of the uniocular image, and therefore the convergence required for fusion, would be the same in both cases. But as a matter of fact a plus cylinder of 1 D, axis vertical, produces about the same amount of binocular metamorphopsia as a plus 2 spherical. This paradox, which I confess puzzled me for some time, I shall explain presently.

Group 3. *Effects of cylinders with axes horizontal.* The fused image appears lower on the side corresponding to the more convex or less concave lens. Let us look at the simplest example which is typical of all the others and in which one

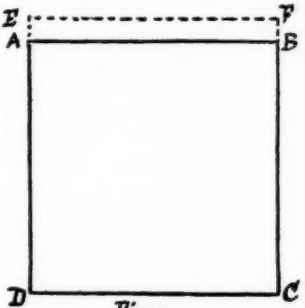

Fig. 4

eye, say the left, is fitted with a plus cylinder, axis horizontal, and the other with a plano. The uniocular effect of plus cylinders with axes horizontal is to increase the height without, apparently, changing the width of the image. Hence we might conclude that the images presented for fusion in this case might be represented by Fig. 4 in which E F C D would be the image seen by the left eye and A B C D that seen by the right. If this were the case the convergence required to fuse the right sides of the two images would be the same as that required to fuse the left sides, and hence the two sides of the fused image ought to be seen at the same distance and therefore they should appear to be the same height, which, as matter of fact, is not true. What is the explanation of this apparent contradiction? First, we must bear in mind that it is the projected images that we fuse in binocular vision. Then I have said (rule 2) that convex glasses make the object

appear nearer. I have also said (rule 3) that when an object seems nearer than it really is it appears smaller. Therefore

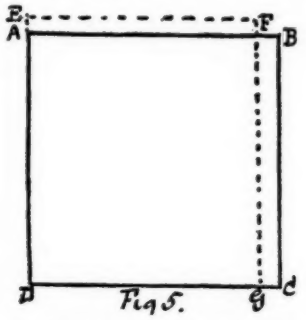

Fig 5.

the images actually projected, one of which requires a subconscious calculation for its formation, are not correctly represented by Fig. 4, but rather by Fig. 5, in which E F G D, the image present to the left eye, is, owing to the causes just stated, somewhat lessened in all its dimensions *including the width*. Here again we have a condition in which the convergence is different on the two sides — less, as shown in Fig. 5, on the right than on the left, making the right side of the fused image (rule 1) seem more distant, and therefore (rule 3) higher, than the other side.

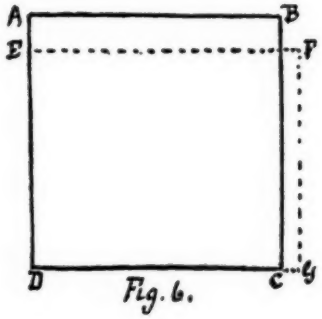

Fig. 6.

Similarly, if we place a minus cylinder, axis horizontal, on one side, say the right, and a plano on the other, we have the condition represented in Fig. 6 in which A B C D stands for

the image seen by the left eye and E F G D that seen by the right. The latter, in accordance with rule 2, appears more remote than it really is and therefore (rule 3) is increased in all its dimensions including the width. It can be seen from Fig. 6 that the convergence is less (divergence greater) for the right sides of the two images than for the left sides. Hence the right side of the fused image appears more distant and therefore higher. It may be added that if diagrams similar to the images in Figs. 5 and 6 be fused by means of a stereoscope they will show the same result, viz., a trapezoid with that side lower which corresponds to the more convex glass, while with diagrams similar to the images in Fig. 4 the result is negative.

We can now understand why a plus spherical lens does not produce as much distortion of the binocular image as a plus cylinder, axis vertical, of the same strength. A plus spherical of 1 D may be considered as the equivalent of two plus cylinders of 1 D, one having its axis vertical and the other horizontal. But we have seen that a plus cylinder with axis horizontal lessens the height of the corresponding side of the square. In point of fact it lessens it so much that a + 1 D spherical has just about half the distorting effect produced by a + 1 cyl. axis vertical. Furthermore, if we place a + 1 cyl. vertical before each eye there will be no binocular metamorphopsia. If now we convert the + 1 cyl. before the left eye into the equivalent of a + 1 spherical by adding to it + 1 cyl. horizontal, the right side of the square at once appears more distant and higher than the left side, just as would happen if we removed both vertical cylinders and replaced them with a + 1 cyl. hor. before the left eye.

The binocular distortion produced by cylinders with axes horizontal is not so promptly observable as when the axes are vertical. A certain time is required for the subconscious calculation to which I have referred and which is involved in determining the dimensions of the uniocular image before comparing it with that of the other eye. This preliminary period varies in different persons and constitutes, in my opinion, a measure of the degree of perfection of the faculty of binocular vision. For, in regard to this faculty, as in respect to the perception of color, we cannot draw a sharp line of

demarcation between "the sheep and the goats." Moreover the metamorphopsia caused by cylinders with axes horizontal is much less pronounced than when the axes are vertical. The proportion is about 1.25 to 3—*i.e.*, a + 1.25 cyl. ax. vert. produces about the same degree of distortion as a − 3 cyl. ax. hor. So that if we place a + 1.25 cyl. ax. vert. before one eye and a − 3 cyl. ax. hor. before the other, a rectangle, as seen, remains rectangular because the projected images have the same width. With this proportion in mind we can predicate the effect of any sphero-cylindrical combination, in which the axes are either vertical or horizontal, on the apparent forms of objects. In this connection we are struck with a phenomenon which at first sight appears paradoxical but which is confirmed by experiment. To put the proportion just given in the form of an equation, + 1.25 cyl. vert. = − 3 cyl. hor. Hence, + 1.25 cyl. vert. + 3 cyl. hor. = 0—*i.e.*, if we place a + 1.25 cyl. vert. ⊖ + 3 cyl. hor. (which is equivalent to + 1.25 sph. ⊖ + 1.75 cyl. hor.) before one eye and nothing before the other, the combined cylinder neutralizes itself as to its effect on the width of the uniocular image and we have no binocular metamorphopsia—a rectangle continues to appear rectangular. Needless to add we have the same result with a − 1.25 sph. ⊖ − 1.75 cyl. hor. before one eye and nothing before the other.

Group 4. *Effects of cylinders with axes oblique.* With a plus cylinder the axis of which is directed upward and inward (toward the nose), before one or before each eye, a square assumes the form of either a trapezium or a trapezoid, wider at the top than at the bottom. If the axes lean outward at the top, the binocular image is narrower above. In the case of concave cylinders the conditions are reversed. If the axes lean upward and outward, the square seems wider at the top; if upward and inward, narrower.

When we look with one eye at a vertical line through a cylinder with axis oblique, the line assumes an oblique position, the obliquity being similar in direction to that of the axis of the cylinder if the latter is concave, and in the contrary direction if the cylinder is convex. I showed in my first paper that this phenomenon is due to the fact that a cylindrical glass may be considered as consisting of two prisms

placed base to base in the line of the axis in the case of convex cylinders, and apex to apex in the case of concave cylinders. When, therefore, we look, say with the right eye, at a vertical line through a convex cylinder whose axis leans upward and to the left, the rays from the upper part of the line pass through a prism, base inward, and are refracted to the left, and are then of course projected to the right, while those from the lower part of the line pass through a prism, base outward, and are refracted to the right and projected to the left. The line, therefore, seems tilted outward at the top—*i.e.*, away from the axis. Concave cylinders, on account of the position of their constituent prisms, tilt the line in a direction similar to that of the axis. The effect upon the two sides of a square

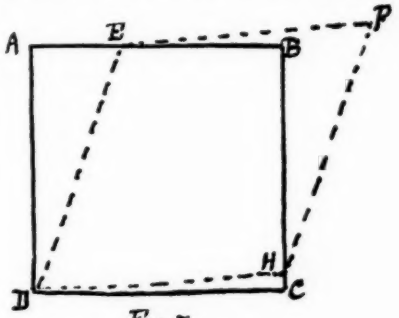

Fig. 7.

is the same as upon the vertical line. Hence, on looking with one eye at a square through a convex cylinder, axis upward and inward, the square is converted into a rhombus leaning upward and outward. If now we look at the square with both eyes, the right armed with a + cyl. axis upward and inward, and the left with a plano, the images projected are as shown in Fig. 7, that of the left eye represented by A B C D, and that of the right by E F H D. It is evident that the eyes are in a state of greater divergence for the upper portions of the respective images than for the lower portions. Therefore (rule 1) the upper part of the fused image seems more distant, and consequently (rule 3) wider, than the lower. If both eyes are furnished with plus cylinders, axes upward and inward, making both uniocular images lean outward at the

top, the effect in widening the upper part of the binocular image is intensified because the excess of the divergence for the upper over that for the lower parts of the uniocular images is increased. If the axis of the cylinder leans outward at the top, so that the position of the prismatic elements, as to their effect upon the rays coming respectively from the top and bottom of the square, is reversed, the result is to make the binocular image narrower at the top. In the case of concave cylinders, for the same reasons, if the axes diverge upward, the square looks wider at the top; if they converge upward, narrower. As we have already observed, the binocular image of a square distorted by a cylinder with axis vertical or horizontal takes the form of a trapezoid with the sides parallel and the upper and lower borders non-parallel. When the axis is at 45° or 135° the image is also a trapezoid with the sides non-parallel and the other borders parallel. In all other positions of the axis the distorted image is a trapezium (no two sides parallel), which approaches the first type of trapezoid as the axis nears the vertical or horizontal, and the second type as the axis nears 45° or 135°. In slowly rotating the axis from vertical to horizontal the different types of distortion melt insensibly into one another, showing that the same laws govern the production of all of them. With cylinders on both sides, of the same strength and with axes parallel, the image may be distorted, but this is uniocular, not binocular, metamorphopsia.

Group 5. *Effects of prisms on the outlines of objects.* If we look at a square card with a prism, say of 8°, base outward, before one eye, and nothing before the other, the side of the card corresponding to the eye with the prism appears higher than the other side. If the prism is held base inward the corresponding side appears lower.

Maddox[1] has shown that, in looking with one eye at a square through a prism tilted so that the base is more distant from the eye than the refracting edge, the dimension of the square corresponding in direction with the *base-apex line*[2]

[1] *The Clinical Use of Prisms*, 1907, p. 132.

[2] "An imaginary straight line from the apex to the center of the base," Maddox, *loc. cit.*, p. 8. This line is of course perpendicular to the refracting edge.

appears shortened—the square looks narrower, and if the refracting edge is more distant the square seems wider. He correctly explains this phenomenon as being the result of the differing angles at which the rays from the different parts of the square are incident. The same result follows even if the *plane*[1] of the prism is parallel to the plane of the face. In looking with the right eye at a square held directly in front of the face through a prism base outward, while the whole square is apparently moved to the left, the left side is moved more than the right side since the rays from the left side, falling more obliquely upon the prism, are more strongly refracted than those from the right side, the result being that the square seems wider. If we uncover the left—unarmed—eye the images to be fused are of unequal width. We have the same condition as is produced by a plus cylinder axis vertical on one side and nothing on the other (represented by Fig. 1) and for the same reasons the fused image appears higher on the right side. If the prism is held base inward the image of the right eye is narrower than that of the left because the rays most strongly refracted come from the left side of the square, so that this side is displaced in the direction of the refracting edge more than the right side. The effect is the same as if a concave cylinder, axis vertical, took the place of the prism, and consequently the fused image appears narrower (lower) on the right side than on the left.

Group 6. *Effects of prisms on the appearance of surfaces.* The apparent depression produced by prisms, bases outward, and the bulging by prisms, bases inward, have long been known. As I remarked at the beginning of this paper the explanations of these phenomena which I have seen—those of Wadsworth,[2] Maddox,[3] and Nagel[4]—are not, in my judgment, entirely satisfactory. Wadsworth's, which is very ingenious, has, I think, the defect of regarding the subject from a purely mathematical (and therefore mechanical)

[1] "The plane, common to the edge and the axis, and which therefore bisects the angle of the prism, and lies equidistant from its two plane faces, is called the *plane* of the prism." Maddox, *loc. cit.*, p. 133.

[2] *Trans. Amer. Ophth. Soc.*, 1883, p. 481.

[3] *The Clinical Use of Prisms*, 1907, p. 138.

[4] Graefe-Saemisch, *Handbuch*, 1880, B. vi., S. 365.

instead of from a physiological (and therefore dynamic) point of view. Maddox suggests the general principle underlying the phenomena, but stops with the suggestion. Nagel's account appears to me to be correct so far as it goes, but it is too incomplete to be regarded as an adequate explanation.

The salient characteristics of the appearances in question are as follows. The outline of the distorted area is more restricted than that of the binocular field, the contraction being most pronounced at the inferior and lateral boundaries. Both pit and protuberance are irregularly spheroidal in form. The deepest part of the pit—or, to speak more accurately, the part most remote from the face—is at the point of fixation. In reality the deepest portion is below this point because the lower margin appears nearer the plane of the face than the upper margin—as if we were looking at the inside of a large bowl whose lower edge is tilted somewhat toward us, with the point of regard, not at the center of the bowl, but a little above the center. The curvature of the pit seems pretty uniform in the horizontal meridian. In the vertical meridian it is not uniform, being less pronounced near the point of fixation than toward the periphery. The hump, seen with abduction prisms, is less clearly defined than the pit seen with adduction prisms. The most prominent part of the protuberance is at the fixation point and the lower edge of the distorted area seems more distant than the upper edge. It remains to be said that the distortions of form under consideration are produced either by one prism or by two. In the latter case the phenomena are merely intensified.

In order to account for this, as for other manifestations of binocular metamorphopsia, it is necessary to determine the forms of the uniocular images. A long vertical line seen with one eye through a prism whose axis is vertical, appears curved, with the concavity facing in the direction of the apex—not exactly in this direction but also slightly upward, so that the chord of the curve inclines toward the base of the prism at the top. This peculiar curvature, which is very noticeable with a strong prism, *e.g.* of 12° and at a distance of three or four feet from the line, is not uniform. Its radius progressively shortens from the point of fixation toward the extremities. The curve is symmetrical for equal distances above and below

the point of fixation. The causation of the curved appearance of the line will be considered presently. The inclined position of the curved line is explained by the fact that the fixation point is not in the center of the visual field but some degrees above it. For this reason the lower end of the vertical line is, from the operation of the causes which produce the curvature, displaced more decidedly in the direction of the apex than the upper end.

Let us now look at a vertical line on a wall with both eyes, one of which—say the right—is furnished with a prism base outward and the other with a plano. The images projected will be as shown in Fig. 8 in which A F B stands for the image of the right eye and C F D for that of the left, the fixed point being at F. It is evident that the eyes are in a state of greater convergence for the extremities than for the point fixed, because, while both eyes are looking at F, the right sees A and B, and the left C and D. Hence (rule 1) the extremities appear nearer than the point of fixation. Therefore the line fused in binocular vision assumes a curved form with the concavity toward the observer. Furthermore, on account of the inclined position of the curved line (seen with one eye), the convergence for the lower extremities is greater than for the upper. Hence the lower end of the fused image seems nearer than the upper end. Now, as Nagel observes, every surface may be regarded as made up of a multitude of parallel lines. The action of the prism on the vertical lines accounts for the concavity from above downward. But the depression is not cylindrical in form but spheroidal. The curvature in a horizontal direction is due, as shown in Fig. 9, to the fact that the vertical measurements of the binocular field gradually diminish toward the lateral boundaries, so that the distance between the extremities of each pair of uniocular images R and L—and, with it, the difference between the convergence for the centers and that for the extremities—progressively decreases until, at the lateral boundaries, it reaches zero. The tilting of the lower margin of the pit toward the observer is explained by the leaning position of the uniocular image

Fig. 8

seen through the prism, which renders the convergence required to fuse the lower extremities of the uniocular images

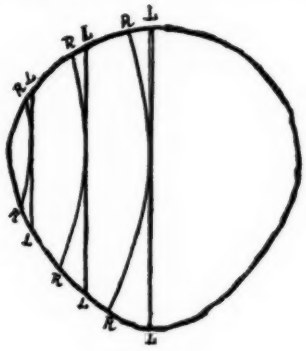

Fig. 9

greater than that required to fuse the upper extremities. That the deepest (most distant) part of the pit is at the fixed point results from the fact that at this point we have the minimum of convergence. The more decided curvature of the pit toward the extremities of the vertical meridian is due to the progressively increasing curvature of the uniocular image from the point of fixation toward the extremities, necessitating a progressively increasing convergence. The effect of two adducting prisms is shown in Fig. 10 in which A F B represents the image projected by the right eye and C F D that by the left. If the prisms are of the same strength the degree of the distortion is of course doubled.

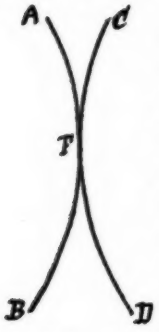

Fig. 10

Theoretically the pit, as we have seen, is formed by the periphery of the binocular field approaching, not by its center receding. Even the bottom of the pit, on account of the convergence caused by the prism at the point of minimum deviation, ought to be seen nearer than the actual wall, and experience confirms theory, but careful observation is necessary on account of our knowledge of the

real position of the wall — knowledge, moreover, buttressed by the uniocular testimony outside of the binocular field.

The depth of the pit at the point of fixation, or at any other point on the vertical meridian, can be measured by ascertaining and then comparing the convergence for such point and that for the upper or lower margin. For this purpose it is necessary to determine the curvature of the vertical line seen uniocularly through the prism. The primary, though not, in my opinion, the only factor in the production of the curvature, is the greater deviation of the rays—projected on the plane of a principal section—which fall obliquely upon the prism from above and below the point of fixation, as compared with the deviation of the rays proceeding from this point. The problem of determining mathematically the deviation of the oblique rays I must leave to some one with more knowledge of geometrical optics than I possess, but approximately accurate results may perhaps be reached in another way as suggested by the following experiment. If we look at a point on a wall, on a level with the eyes, through a prism with its refracting edge vertical and its "plane" parallel to the wall, the point appears displaced in the direction of the apex in proportion to the strength of the prism and to its distance from the point. If now we rotate the prism on its "base-apex line," tilting the upper border away from the face, the displacement of the point is increased, slowly at first, but with increasing rapidity as the rotation proceeds. When the plane of the prism approaches the horizontal—*i.e.*, just before the critical angle is reached, the displacing power of the prism is about doubled.[1]

By means of a simple apparatus constructed for the purpose

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[1] Dr. Drualt, formerly of Paris, now of Tours, finds by calculation that at the critical angle "la déviation est un peu plus que doublée. Soit, par exemple, un prisme de 3° entre les faces, avec indice 1.52, sa déviation *minima* est 1°.33′.38″. Si le rayon incident a une direction primitive perpendiculare à la face postérieure, la déviation n'est augmentée que de quelques secondes: 1°.33′.44″; elle est *latérale* si l'arrête du prisme est verticale. Si ce prisme tourne alors de 90°, de façon que sa face postérieure devienne horizontale et son arrête parallèle au rayon incident, celui-ci subit une déviation *latérale* de 3°.25′.42″, plus une déviation en bas."

and which indicates the inclination of the prism plane from the vertical, I have observed the horizontal displacements produced at different inclinations, and from them computed the deviation of a prism No. 3 (1½ d.) for each inclination. The results, so far as they affect our present purpose, appear in the following table. The increase in the displacement up to an inclination of 20′ is very slight. Even at 20° it is only $\frac{1}{16}$ of the minimum displacement.

Table of Deviations.

Incl'n of prism plane from the vertical.	Deviation of oblique rays.	Incl'n of prism plane from the vertical.	Deviation of oblique rays.
Vertical	1°.30′	50°	1°.50′.17″
20°	1°.33′.27″	53°	1°.52′.46″
25°	1°.34′.42″	55°	1°.55′.12″
30°	1°.36′.36″	60°	2°. 1′.30″
35°	1°.39′	65°	2°. 8′.24″
40°	1°.42′	70°	2°.16′.30″
45°	1°.45′.54″	75°	2°.25′.12″

Needless to say the effect is the same whether the prism plane be regarded as inclined to the incident ray, the latter being horizontal, or the ray to the prism plane, which remains vertical. In the latter case the angles of inclinations count from the horizontal and thus correspond to the angles which represent vertical measurements of the visual field. Thus if the height of the field is 50° the deviation of the rays which come from the top of the field and are projected on the plane of a principal section of a prism No. 3 (1½ d.), is 1°.50′.17″.

In Fig. 11 B C represents the upper part of the vertical line seen through the prism P at A, B the point of fixation, A the angle which measures the height of the visual field, and *b* and *c* rays coming respectively from C and B. Let β be the deviation of ray *c*, which is of course projected on the plane of a principal section, and β_1, the deviation of ray *b* projected on a similar plane. Now the curvature, as we see it, is notably greater than would be accounted for by the deviation β_1, if the table of deviations given above is anything like correct.

The explanation is that the displacement of any given point on the vertical line depends, not only on the deviation, but also on the distance at which it is projected. Hence in comparing the displacement of the point C with that of B, the former corresponds, not to β_1, but to $\beta_1 \frac{b}{c}$ (1) which we shall call β_2. But $\frac{c}{b} = \cos.\ A$. Therefore $\frac{b}{c} = \frac{1}{\cos.\ A}$. Therefore $\beta_2 = \frac{\beta_1}{\cos.\ A.}$ (2). As the prisms used in ophthalmology are generally weak, and β_1 and β_2 are small angles, we may, with a slight margin of error, substitute the sines for the angles, so that the formula becomes $\sin.\ \beta_2 = \frac{\sin.\beta_1}{\cos.\ A}$ (3). For strong prisms, and to ensure perfect accuracy in using any prism, the formula $\beta_2 = \beta_1 \frac{b}{c}$ is required. The rationale of this formula is a

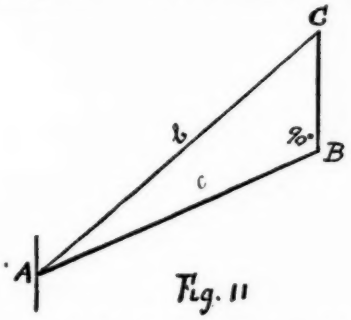


Fig. 11

matter of physiological rather than geometrical optics and may be stated thus: The curved line appears to lie, like the original line, in a vertical plane. Hence, while the causes which produce the curvature are operative at differing distances (*b* and c), the effect of these causes is referred to one and the same distance. That is to say, the displacement proportionate to the distance *b* is unconsciously applied to the distance *c* and is therefore attributed to a deviation which is correspondingly greater than the actual deviation and which may be called the assumed deviation.

To measure the depth of the pit under consideration the data required are the minimum deviation of the prism (β), the distance between the prism and the wall, the height of the binocular field, and the distance between the centers of ocular rotation. For the latter we may substitute the interpupillary distance without sensibly affecting the result. Let us suppose

the minimum deviation of the prism (β) to be 1°. 30′, the distance from prism to wall to be 50*cm*, and, as in my case, the interpupillary distance to be 6.4*cm* and the height of the binocular field 53°. In the table of deviations given above, the angle β_1, corresponding to a visual field angle of 53°, is 1°.52′.46″. Following formula (3), sin. $\beta_2 = \frac{\text{sin. } 1^\circ.52'.46''}{\text{cos. } 58^\circ}$ Hence β_2, the assumed deviation, is 3°.7′.24″.

In Fig. 12, which is intended to show the effect of an adducting prism of 1½ d. before each eye at a distance of 50*cm* from prism to wall, P and P′ represent the pupils, 6.4*cm* apart,

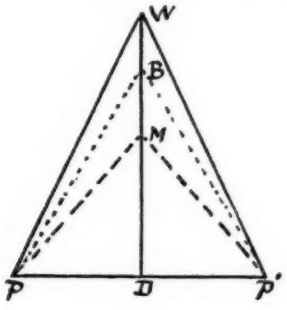


Fig. 12

W the wall, B the bottom of the pit, and M its upper margin. Angle W P B represents the ocular deflection produced by a prism with a deviation of 1°.30′ and W P M that produced by a prism with a deviation of 3°.7′.24″.

W P B 1°.25′.30″.[1]

W P M 2°.58′.2″.[1]

W P D (whose tan. $= \frac{5.0}{3.3}$ = 86°.20′.17″.

Hence B P D (= W P D — W P B) = 84°.54′.47″.

and M P D (= W P D — W P M) = 83°.22′.15″

D B = P D tan. B P D = 35.95*cm*.

D M = P D tan. M P D = 27.53*cm*.

Hence M B, the depth of the pit, = 8.42*cm* or 3⅓ inches,

[1] Calculated by the rule laid down by Maddox, who has shown that in near vision the ocular deflection caused by a prism is less than the light deviation, *loc. cit.*, p. 178.

which accords very well with my estimate of the depth of the pit produced by a prism of 1½ d., apex inward, before each eye.

The protuberance caused by looking through prisms with bases inward is explained on the same principle. We may make use of Fig. 10 to illustrate what takes place. But now C F D represents the image of the right, and A F D that of the left eye. Clearly the eyes are more divergent for A and C and for B and D than they are for F, so that the upper and lower borders appear more distant than the point of fixation. Moreover, the divergence is greater for the lower border than for the upper. Hence the former appears more distant than the latter. The distorting effect of abduction prisms is not usually so obvious as that of adduction prisms. This difference is probably, in part at least, due to the fact that the former are in general more difficult to overcome; and the estimate of the height of the hump is not so easily made, because, with prisms, bases inward, on account of the approach to parallelism of the visual axes, the conditions are similar to those which obtain in estimating relative distances which are remote.

It is scarcely necessary to say that, although the binocular metamorphopsia under discussion is more strikingly manifested in looking at flat surfaces, the appearance of all opaque surfaces is modified in the same way. In looking with prisms into a mirror, the latter remains flat, especially if it is perfectly polished, but the reflected image is changed in form, so that we may at will add to the prominence of a too modest chest, or repress the exuberance of a too assertive abdomen.

From the foregoing analysis of the phenomena concerned in the production of binocular metamorphopsia it is, I think, clear that these phenomena are adequately explained on the two assumptions with which we started out, viz.: that the projected images are those which are fused in binocular vision, and that convergence is the all-important factor in such fusion. In the light of this analysis we are able to formulate the general principle underlying the distortion of the binocular image. In fixing a given point of any surface, whether presenting relief or not, there is a definite relation between the direct or fixing convergence and the indirect convergence—*i.e.* the convergence for those points in the uniocular projected images which are outside the point of fixation. *E.g.*,

in looking at an object shaped like a section of a sphere with the pole facing us, if we fix the pole, the convergence is greatest at that point and diminishes gradually toward the periphery. If we fix a point on the periphery, the convergence is least at the point of fixation and increases gradually toward the pole. But, whatever the point fixed, the relation between the direct and the indirect convergence is constant, so that two or more competent observers will agree as to the form and position of the object. If now we place a plus cylinder, axis vertical, before the right eye and fix the pole, the convergence for the latter will be unchanged, while that for the other points will be altered, diminishing steadily toward the right side and increasing toward the left side, and thus making the latter appear nearer, and the former more distant, than before, and changing the apparent form of the object. If, instead of a cylinder, we place a prism, apex inward, before one or before each eye, while the convergence for the whole object is increased, that for the periphery will be increased more than that for the pole, so that the spherical surface will seem flatter. We may therefore describe the condition giving rise to binocular metamorphopsia *as a disturbance in the relation between the direct and the indirect convergence.* This disturbance arises from a certain dissimilarity between the uniocular images. The dissimilarity may take one or both of two forms: 1st, inequality in width, as produced by spherical glasses or by cylinders with axes vertical or horizontal; 2d, want of parallelism between the images of the vertical lines which help to make up every surface. The want of parallelism may show itself as either a difference in the angle of inclination, the effect of cylinders with axes oblique, or a difference in form (curvature), the effect of prisms. These conditions constitute the essential points referred to in the simile of a judicial trial used on a preceding page. Any one of them renders impossible such a superposition of the projected images as will make convergence for the corresponding points of these images normal at any given instant.

We have so far studied the phenomena of binocular metamorphopsia as regards their production. We have now to consider them from another point of view. I pointed out in my first paper that, if the glasses which cause the distortion

are worn constantly for a period which varies with different persons, objects gradually assume their normal form. *E.g.*, a right-sided hypermetrope, who has been given the correcting glass which at first made the right side of a square appear higher than the left side, learns in time to see the square correctly. When this result is achieved, if the glass is removed the distortion reappears with the left side wider. And if the constant wearing of the glass is now discontinued the distortion once more disappears—this time more rapidly than before. *Chassez la nature, elle revient au galop.*

How is the change from binocular metamorphopsia to binocular orthomorphopsia brought about? The answer to this question, in my opinion, involves a choice between the two conflicting theories of vision usually styled nativistic and empiristic. According to the first, each of the retinal percipient units corresponds to a definite direction in space in relation with certain fixed planes of the head. The perception of direction is therefore a purely retinal function and may be properly placed among the primary sense perceptions. The retino-spatial correspondence may be primordial, *i.e.* racial, or it may be the result of transmitted experience. In either case it is fixed at birth and unchangeable. Hence the projected images must correspond in form and, relatively, in size with the retinal images, but it is the latter which are fused in binocular vision. Each point in one retina has its corresponding point in the other, and this interretinal correspondence, like the retino-spatial correspondence, is based on anatomy and therefore unchangeable.

According to the empiristic conception, projection is not a function of the retina but of the subconscious brain centers. The perception of direction is distinctly not a primary sense perception but a judgment based on data furnished, in the first instance, by the innervational apparatus of the ocular muscles. The projected images may be, and perhaps usually are, dissimilar to the retinal images, and it is the former that are fused in binocular vision. An extreme form of the empiristic theory is that at birth the retina, or rather the visual apparatus, is, so far as projection is concerned, a blank page, to be filled by the lessons of experience. A more moderate form is that the faculty of direction is more or less highly developed at

birth as the result of transmitted experience, but that the transmitted experience of the race can be modified by the acquired experience of the individual. As between these two forms of the empiristic theory, the latter seems to be the better sustained by the teachings of comparative physiology. Inasmuch as one form of the empiristic theory is also nativistic, the latter designation might be substituted by the term anatomical.

Which of these two conceptions, the anatomical or the empiristic, offers the better basis for an explanation of the conversion of binocular metamorphopsia into binocular orthomorphopsia? We have seen that the various examples of binocular distortion described in this paper are satisfactorily accounted for by the dissimilarity between the projected uniocular images, and that the essential feature of this dissimilarity relates to the degrees of convergence necessary for the different parts of these images. It seems a logical conclusion that orthomorphopsia is regained by the disappearance of this dissimilarity. Now according to the anatomical theory the retinal and the projected images are necessarily similar in form. Therefore upon this theory the unequal images formed on the retinæ by a plus spherical glass on one side and nothing on the other would have in some manner to become equal, at least in width, in order that the binocular distortion should disappear.

Let us examine this question in the light of one of the simplest concrete examples, viz: a case of one-sided simple hypermetropic astigmatism, axis vertical; and, in the interests of logical sequence, let us "begin at the beginning." I have previously shown that ametropic persons may be regarded as in the same condition, optically, as emmetropes compelled to wear glasses corresponding to the ametropia. Hence a case of left-sided hypermetropic astigmatism, axis vertical, would present the condition illustrated by Fig. 1, and therefore there must have been binocular metamorphopsia when the eyes were first used in binocular vision. But there is no metamorphopsia now: The retinal images, on the anatomical theory, have become equally wide. Is such a change possible? Theoretically the narrower image of the astigmatic eye could be made like that of its fellow by such an accommodative

effort as would produce a neutralizing astigmatism of the lens. But, as I showed in my former paper, the act of accommodation is not concerned in the production of binocular metamorphopsia. Besides, all of the phenomena under discussion, including the regaining of orthomorphopsia, are witnessed after the extraction of cataract on both sides. And the extrinsic, equally with the intrinsic, muscles may be excluded from consideration in this connection because no possible movements of the eyeballs could have the effect of making the retinal images at a given instant equal in width. There is in my judgment no conceivable way in which this result could be attained.

The binocular distortion produced by cylindrical glasses with axes oblique has attracted much more attention than other forms of optical metamorphopsia. Savage,[1] to whom we are indebted for so many valuable contributions to our knowledge of the diagnosis and treatment of muscular affections, and who is a pronounced advocate of the anatomical theory of projection, explains the disappearance of the distortion due to oblique astigmatism by a torsional heterotropia the rationale of which would be as follows: Horizontal and vertical lines in space are perceived, respectively, as such by normally horizontal and vertical rows of retinal units. In a case of oblique astigmatism of one eye, the other being normal, when the rays from horizontal lines in space enter the astigmatic eye they fall, owing to the prismatic twist of the refractive defect, obliquely across the normally (and actually) horizontal rows of retinal units. Or as the author correctly says, "the retinal image is displaced toward the meridian of greatest curvature."[2] Hence the horizontal lines appear oblique and there is binocular metamorphopsia because the two retinal images do not correspond. To overcome the obliquity, the astigmatic eye gradually rotates on its anteroposterior axis to an abnormal position (cyclotropia), the effect of which is to make the horizontal lines assume their true position, and the metamorphopsia disappears. When the correcting lens is applied, the distortion is again produced on account of the cyclotropia. The latter is gradually overcome

[1] *Ophthalmic Myology*, 1911, p. 417 *et seq.*
[2] *Ophthalmic Record*, vol. i. (1891), p. 9.

and orthomorphopsia is once more established. I am inclined to doubt the truth of this hypothesis because, in the first place, it has not, so far as I am aware, been objectively demonstrated. Secondly, whatever may happen in binocular vision, there is admittedly no cyclotropia in uniocular vision, and the permanently correct axis of astigmatism is therefore determinable at once. Hence, after correction, the combined vision of the two eyes would be relatively imperfect until the disappearance of the cyclotropia, whereas, as a matter of fact, according to my experience, the best combined vision is obtained immediately. Thirdly, we have seen that the same laws govern the production of all of the forms of binocular metamorphopsia produced by cylinders whatever the position of the axis. We have also seen that when the axis is vertical or horizontal the distortion disappears without the influence of direct muscular interference. It is then highly improbable that such interference is a factor in the disappearance of the distortion produced when the axis is oblique. Fourthly, the cyclotropia hypothesis is not needed to explain the phenomena. The changes from orthomorphopsia to metamorphopsia and back again are observed in vision with one eye, in which case, as just now said, there is no cyclotropia. *E.g.*, an uncorrected anisometrope, with one eye normal and the other obliquely astigmatic, sees a square as perfect in form with the astigmatic eye as with the other. After correction the square appears distorted to the corrected eye. Then, after the lapse of a certain time during which the glass is worn constantly, the uniocular metamorphopsia disappears. Now uniocular metamorphopsia is the essential prerequisite of binocular metamorphopsia, and with the disappearance of the former the latter also disappears. But even if we were to admit the existence of cyclotropia in these cases it is clear that the torsion, which, in an obliquely astigmatic eye, would cause the rays from horizontal lines to fall upon normally horizontal rows of retinal units, would not at the same time permit the rays from vertical lines to fall upon normally vertical rows of units. Such torsion, therefore, could not remove the cause of the metamorphopsia, viz., the disturbance of the relation between the direct and the indirect convergence. Is there then no action of the oblique

muscles in these cases? Undoubtedly there is, but this action, like that of the other ocular muscles, manifests itself in connection with the educational process which will be spoken of presently, and by which correct projection is attained; and the extra strain put upon the obliques under these circumstances would sufficiently account for the development of symptoms incident to the condition which Price[1] happily termed cyclophoria, and which Savage[2] was the first to describe. The change in the appearance of surfaces caused by prisms, like other forms of binocular metamorphopsia, is corrected when the prisms are worn for some time, because the vertical lines which help to constitute the surface, and which are at first seen curved, gradually come to appear as they really are; but there is no conceivable means by which the images of these lines could be made other than curved on the retina. Finally, as I have already indicated, we may have marked differences in the form and size of the retinal images without binocular metamorphopsia resulting, as, for example, with a $\pm$ 1.25 $\bigcirc$ $\pm$ 1.75 cyl., or any proportionate combination, before one eye and nothing before the other; whereas if we change the proportion of the spherical to the cylindrical element of the glass binocular distortion is immediately observed. The foregoing considerations lead, I think, to the conclusion that the anatomical view of projection cannot be reconciled with the facts of binocular metamorphopsia. Nor does such a reconciliation seem capable of being effected by Javal's theory, that in binocular vision one of the uniocular images is suppressed, because the binocular image in these cases may bear no resemblance to either of the retinal images.

From the empiristic standpoint false or imperfect projection can be rectified through an educational process during which the mind, as it were, feels its way by an incessant action —*tâtonnement*—of the ocular muscles in changing the point of regard from one part of the visual field to another. But, as everybody realizes, the perception of form is instantaneous, the explanation being that, when the educational process is

[1] Quoted by Savage, *Ophthalmic Myology*, p. 383.
[2] ARCH. OF OPHTH., January, 1891.

completed, the muscular movements are no longer necessary, although they may aid in confirming or correcting the first impression. The retinal units have, as a result of a multitude of experiments, found their places in the retinal system, *i.e.* their relation to the yellow spot and to each other; and this relation, once established, remains, through a species of subconscious memory, fixed, until some disturbing influence intervenes. With the advent of such disturbing influence, in the shape of correcting glasses for an existing anisometropia, the educational process has to begin over again and to be continued until correct projection is attained. According to this view the retina is maid, not mistress, and requires training. We are not the slaves of our retinæ any more than the skilled marksman is a slave to his imperfect gun the defects of which he has learned to allow for. El Greco's bizarre drawing and Giotto's perfect "O" do not prove that the one artist was astigmatic and the other emmetropic. A straight line is seen as such by a person affected with irregular astigmatism, although the retinal image of the line must be anything but straight, and the false projection induced by prisms is overcome with practice.[1] The results of the investigations of a considerable number of observers and, notably, the facts presented in the interesting paper by Lister and Holmes[2] warrant the belief that we shall some day be in a position to map out, point for point, on the visual area of the cortex, the cerebral analogues of the retinæ. But, even if we are able to locate all of the centers concerned in vision and, in addition, to trace the connecting fibers which serve to correlate their various activities, this knowledge will not suffice for the solution of all visual problems, because the entity, emanation, or whatever we choose to consider it, which we call mind is, in vision, to a certain extent, independent of the visual apparatus, the information conveyed by the latter being tested in the light of previous knowledge. We see, or we can learn to see, objects of the form which we know them to possess.

The empiristic theory is, moreover, corroborated by certain

[1] Tscherning, *Optique Physiologique*, 1898, p. 286.

[2] "Disturbances of Vision from Cerebral Lesions, etc," *Royal Soc. of Med., Section Ophthalmology*, March 22, 1916.

phenomena connected with the sense of touch. The chords which, with bewildering rapidity, the accomplished pianist summons from his instrument, are the result of a sort of tactual projection made possible by laborious training of the muscles of arms, hands, and fingers. Sight is conceded to be only a glorified touch. The mechanism by which the blind boat-builder palpates a block of wood and creates a cup-winner is essentially the same as that by which the astronomer palpates the planets and evolves a solar system.

The binocular image is formed by superposing and comparing the uniocular images, the superposition being effected by the convergence required to permit light-beams from some given point of the object regarded to fall simultaneously, in the interests of single vision, upon the two foveæ. In the formation of the binocular image the underlying principle is the same as that underlying the formation of the uniocular images. Both kinds of images represent judgments based on information supplied, primarily, by the muscular apparatus. From the muscular efforts concerned in projection we know the form—the outline—of the images, but we do not know the distance at which they are seen, and therefore we cannot properly estimate their size. Moreover, we get no adequate notion of relief which depends upon the relative distances at which are seen the several parts of the images. The estimate of distance, which is a cardinal factor in binocular vision, is made possible by convergence. But the experiment of Dove, accepted by Donders,[1] Giraud-Teulon[2] and others, and abundantly confirmed and elaborated by Le Conte,[3] shows that the theory of Bruecke, viz., that in order to obtain the idea of relief, the eyes must fix in turn every point in the visual field, is not rigidly correct. Nevertheless this theory, if not complete, is the first sentence of a complete theory—if not the house it is the most important part of the house—*i.e.*, the foundation. The true explanation seems to be that, just as by a subconscious memory of projection, the outcome of accumulated experience, partly hereditary, partly acquired, we can instantaneously form the uniocular images, so by a

[1] Quoted by Giraud-Teulon, *La Vision et ses Anomalies*, 1881, p. 617.

[2] *Loc. cit.*, p. 617.

[3] *Sight*, 1881, pp. 148–150.

subconscious memory of convergence we know, without actually converging, how much convergence would be required to fix any particular point in the binocular field. In other words we can estimate the direct from the indirect convergence.[1] As binocular metamorphopsia results from a disturbance of the normal relation between the direct and the indirect convergence, orthomorphopsia is restored by the removal of the disturbance through acquiring a correct projection of the uniocular images.

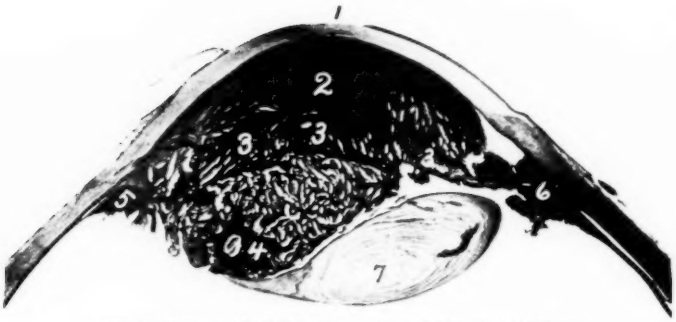
It is not the purpose of this paper to discuss the anatomical and empiristic theories of projection and binocular vision apart from their bearing on the phenomena of binocular metamorphopsia. From a study of these phenomena, especially the spontaneous conversion of metamorphopsia into orthomorphopsia, we are led to the conclusion that retinal physiology bears no constant relation to retinal anatomy; that the directions in which images are projected by means of the same retinal elements are subject to variation, and that, for purposes of vision, the corresponding points in the two retinæ *which count*, are those which correspond in function and not necessarily in structure. The marriage of corresponding retinal points is a loose tie the harmony of which is easily disturbed by interposing new optical conditions, and, as sometimes happens in the social sphere, divorce and remarriage with a more sympathetic partner are effected without undue delay.

[1] This view is similar to, if not identical with, that expressed by Le Conte, *loc. cit.*, p. 151.

ILLUSTRATING DR. BELL'S ARTICLE ON "A CASE OF SARCOMA OF THE IRIS."

Read before the American Ophthalmological Society at Hot Springs, Va.

Primary Sarcoma of the Iris.



Photomicrograph Melano-Sarcoma of Iris, x 6½ Diam.

1.—Cornea with portion of epithelium stripped. 2.—Portion of tumor in anterior chamber. 3.—Remains of iris. 4.—Portion of tumor in posterior chamber (between 3 and lens). 5.—Ciliary body, tumor cells invading the head and processes. 6.—Ciliary body not invaded. 7.—Lens.

REPORT OF A CASE OF PRIMARY SARCOMA OF THE IRIS: WITH REMARKS.[1]

By Dr. GEORGE HUSTON BELL, New York,
OPHTHALMIC SURGEON, NEW YORK EYE AND EAR INFIRMARY.

(With one colored illustration.)

PRIMARY sarcoma of the iris is so seldom seen or reported that I thought the following case would be of interest to the Society.

The patient, a man aged eighty years, came to the New York Eye and Ear Infirmary on October 27, 1915, complaining of pain in his left eye and loss of vision. He gave the following history: Has been a great smoker all his life, but has not used alcohol. Enjoyed excellent health up to twelve years ago, when he had a paralytic stroke; since then he has had poor vision in his left eye. Two years ago he was struck in the left eye with a Roman-candle ball.

On examining the patient I found that there was a dark nodular mass in the upper nasal quadrant of the anterior chamber, apparently between the cornea and iris, and involving the latter. The tumor was *6mm* in length and *3mm* in breadth. The borders were sharply defined, and the neighboring iris tissue was not discolored or hazy. There was an exudate in the pupillary area corresponding to the measurements of a smaller mass, shown in the drawing (Fig. 1). The larger growth was triangular in shape, the base of the triangle being upward and inward. The mass filled the angle of the anterior chamber in the region of the growth. The tension in the eye was 52, showing that he had secondary glaucoma, which always supervenes in the second stage of the disease. The vision of the eye was hand movements. A clinical diagnosis was made at that time, but the patient refused to enter the hospital.

[1] Read at meeting of American Ophthalmological Society, 1917.

He was given a 2 per cent. solution of pilocarpin and requested to return to the clinic. He was next seen on November 26th. The tumor had grown 1*mm* in all directions. The eye was more painful and there was considerable pericorneal injection. Transillumination at this time over the ciliary body gave no shadow. The patient still refused to enter the hospital, but came to the clinic occasionally. Finally, in May, 1916, the tension in the eye got up to 90*mm*, and the pain was so great that he consented to come into the hospital. The eye was enucleated on May 29, 1916, under novocain anæsthesia, on account of the patient having a bad heart and his advanced years. Healing was uneventful.

His right eye showed some arteriosclerotic changes in the fundus, but he still had a vision of $\frac{20}{40}$ with correction and a tension of 20*mm*. The iris was free from any abnormal pigment changes. I have not seen the patient recently, but the last time he was at the clinic he was enjoying good health for a man of his years.

PATHOLOGIC REPORT.—The following is the report of Dr. George S. Dixon, the pathologist.

In the case of Mr. Antonio A., the left eye submitted presented a dark growth occupying nearly the whole of the upper nasal quadrant of the anterior chamber.

Sections through the growth showed that the lens had been dislocated downward, backward, and outward, but the tumor had not invaded the vitreous chamber to any considerable degree; it, however, had attacked the head of the ciliary body and also the ciliary processes, and a few cells had entered the canal of Schlemm, having penetrated at the angle. Descemet's membrane was otherwise intact, but the endothelial lining had disappeared.

The growth was a melanosarcoma, the type of cell being principally spindle. There were quite a number of oat-shaped cells, and the balance were round. There was a considerable amount of pigment, but it was not excessive. The original iris blood-vessels were present in the growth and were in some places widely separated.

There was pronounced retinal œdema, especially in the inner layers. The choroid was atrophic.

Diagnosis: Primary melanosarcoma of the iris.

Fuchs (1) found primary sarcoma of iris 16 times in 259 collected cases of sarcoma of uveal tract. Martin, one in 43; and Lawford and Treacher Collins, once in 103 cases examined at Moorfields.

Wood and Pusey (2), in 1902, gave abstracts of 83 cases in which microscopic examinations were made.

Thorington (3) reported a case in 1910 of leukosarcoma of iris.

Laven (4), in 1913, reported a case of primary sarcoma of iris in a girl twelve years of age, and reviewed the literature of 13 cases. In one of the cases reported by Laven enucleation was done eleven years after the excision of the tumor, and death followed six years later from general metastasis.

This case of mine is the first one to find its way to the laboratory of the New York Eye and Ear Infirmary since May, 1912. One reason for this, I think, is that, as a rule, these tumors are of exceedingly slow growth and it is very difficult to convince patients of the gravity of the disease unless there are marked inflammatory symptoms or the vision is greatly impaired. Some authors (5) claim that they can exist for months or even years without causing vascular injection of the eye or interfering with the motility of the iris. I think it is most important to call the attention of patients to these "suspicious spots," as they may be slow-growing melanosarcomata.

The great question that arises is one of responsibility in these cases of sarcomata of the iris, especially when they are seen in the primary stage of the disease, before iritis or inflammatory symptoms have developed or secondary glaucoma supervenes. Of course, in my case the responsibility was not great, as the tumor was in the secondary stage of the disease when the patient was first seen, and it was not hard to decide what was the best course to pursue. However, it ought not to be a difficult matter for the surgeon in any case of primary sarcoma of the iris, when the diagnosis is once established, although Fuchs (6) says that very small sarcomata of the iris can be removed by iridectomy, those parts of the iris which bear the nodules of the tumor being excised.

How are we to know clinically in a given case whether the sarcoma cells have invaded the ciliary body, ciliary processes, or even the canal of Schlemm?

CONCLUSIONS.

1. I feel that the prognosis is always unfavorable when the sarcoma is excised.

2. Iridectomy should be done only as an aid to diagnosis.

3. I quite agree with Wood and Pusey who, after a thorough investigation of the subject, came to the conclusion that, when the diagnosis of iris sarcoma is established, the globe containing the growth should be immediately enucleated.

4. That they do recur sooner or later when excised and are apt to produce general metastases.

5. I feel that it is not fair to jeopardize the life of the patient by a compromise in the treatment, and, after all is said and done, in my opinion the radical operation is by far the safest in the end.

I wish to express my great appreciation and indebtedness to Dr. George S. Dixon for the excellent photomicrograph illustrating my paper.

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ILLUSTRATING DR. WOODS'S AND DR. DE SCHWEINITZ'S ARTICLE ON "TRYPANOSOME KERATITIS—AN EXPERIMENTAL STUDY."

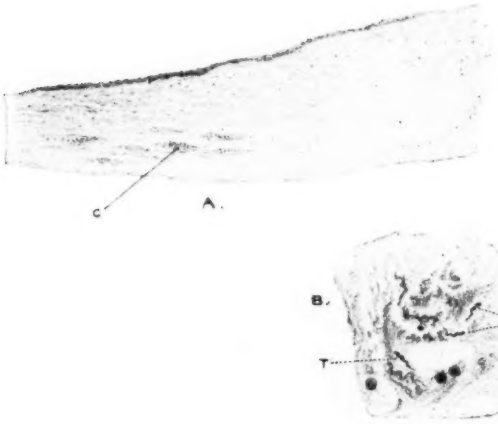

FIG. 1. *A—Cornea stained by H. & E. Low Power.* Showing capillary invasion (*c*), and looseness in substantia propria, with cellular infiltration.

B—Cornea (x 1090) showing actual presence of trypanosomes in lamellæ.

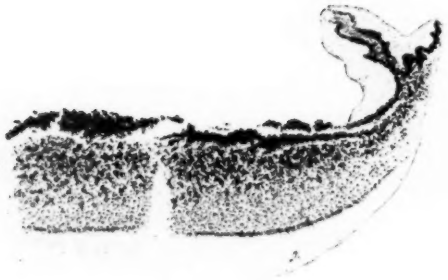

FIG. 2.—Iris and Ciliary Process. *Low Power.* Showing cellular infiltration of iris and exudate around ciliary process.

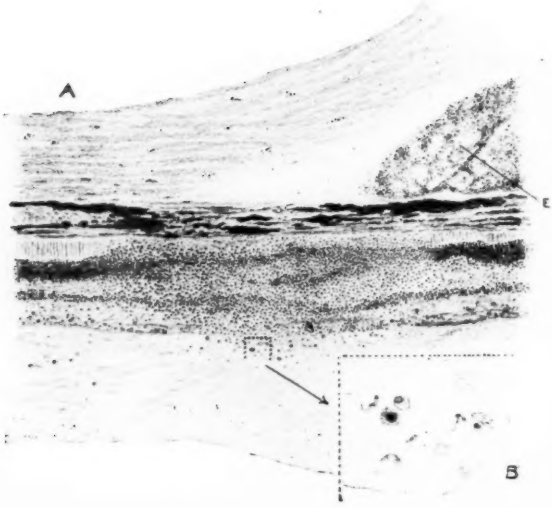

FIG. 3. *A*—Focal Lesion of Retina. Showing disarrangement of retinal layers and mononuclear infiltration. *E*—Sero-fibrinous-cellular exudate between choroid and sclera. *B*—Exudate over retina (x 1090) showing trypanosomes and type of cells.

TRYPANOSOME KERATITIS—AN EXPERIMENTAL STUDY.[1]

By Dr. ALAN C. WOODS and Dr. GEORGE E. DE SCHWEINITZ, Philadelphia.

(With three illustrations on Text-Plate X.)

(From the John Herr Musser Department of Research Medicine, University of Pennsylvania, Philadelphia.)

OUR interest in the lesions here described was aroused through being asked to give an opinion on certain ocular disturbances occurring in the course of the general study of experimental trypanosomiasis in the dog.[2]

The nature of these lesions, in view of our knowledge of the ocular reactions in man, and in view of the observation of Morax (1) and Natten-Larrier and Monthius (2) that such experimental lesions have a striking similarity to those caused by trypanemia of syphilis, led us to undertake an extended investigation of the ocular lesions occurring in the dog as the result of infection by the Trypanosoma equiperdum.

Ocular lesions of trypanosomiasis in man have been described by Morax, Natten-Larrier, Monthius, da Gama Pinto, Laveran and Pettit, Inman, Jellett, and others. The subjective symptoms, namely, ocular æsthesia, trembling of objects, and anæsthesia of the conjunctiva, are well known, as are also the objective symptoms, keratitis, iritis, chorioretinitis, and œdema of the lids. As, however, these disturbances yield readily to trypanocidal treatment, there has been no

[1] Read at meeting of the American Ophthalmological Society, 1917.

[2] Krumbhaar, E. B., and Pearce, R. M.: "Experimental Trypanosomiasis in the Dog," *Jour. Inf. Diseases* (to appear shortly).

opportunity in man for the microscopic study of the details of the pathologic lesions. It was, therefore, in the hope of adding something to our knowledge of the pathology of such ocular lesions that we undertook the study of the experimental lesions in the dog.

HISTORICAL.

Experimental ocular lesions caused by trypanosomes were first recognized by Kanthack, Durham, and Blandford (3) in 1899. These authors observed corneal opacities, conjunctivitis, and turbidity of the aqueous in animals infected with trypanosomes, and were able to demonstrate trypanosomes in the aqueous of such animals. Elmassian and Migone (4), in 1903, described keratitis and iritis in horses infected with Trypanosoma equiperdum, and state that the inoculation of this trypanosome in dogs produced similar symptoms. Laveran and Mesnil (5) in 1904 studied the ocular lesions produced in rabbits by the Trypanosoma nagana. According to these authors, the local symptoms appeared about twenty days after inoculation, and lasted from thirteen to fifty-eight days. The first symptoms were a blepharoconjunctivitis with coryza, which became purulent. The pus collected under the lids; the cornea became secondarily involved and opacified rapidly. In 1906 Stock (6) studied trypanosome ocular lesions and concluded that the corneal opacities resulted from an œdema of the cornea. An iritis sometimes occurred. The lesion in the cornea appeared to be a leucocytic infiltration. Trypanosomes occurred in the aqueous, but did not invade the cornea. The corneal lesion pathologically was an interstitial keratitis.

Stargardt (7) found trypanosomes in the anterior chamber and in the subconjunctival tissue. The trypanosomes, according to Stargardt, appeared in the aqueous on the second or third day of the eye disease, and then disappeared.

Morax (8), in 1907, reported upon the pathologic picture of experimental trypanosome ocular lesions. The lesions occur throughout the entire eye, but more especially in the anterior segment—the cornea and the iris. The corneal lesions are of three varieties. The first type consists in localized areas

of œdema in the substantia propria of the cornea; the lamellæ are separated by a serous exudation, and in this exudation are large numbers of trypanosomes. There is but little cellular infiltration. The second type is similar to the first, except that but few trypanosomes are present in the areas of œdema, while there is a dense infiltration of mononuclear cells and a few leucocytes. The third type is notable on account of the absence of trypanosomes, and the vascularization which may take place. According to this statement, there is an actual invasion of the corneal tissue by trypanosomes, similar to that by the spirochæte in interstitial keratitis. In contrast to this report of Morax are the experiments of Leber, which appeared in the following year. Leber (9) found that a parenchymatous keratitis occurred in rabbits and dogs which had trypanosomes in the circulating blood and in the aqueous. The injection of killed trypanosomes in the anterior chamber caused a keratitis, and Leber, therefore, believed that the ocular lesions were due to a toxin formed by the trypanosomes. Killed trypanosomes injected subconjunctivally caused a keratitis. The cornea of an eye showing a trypanosome keratitis was clipped off, macerated, and injected in a rat, and the rat did not develop trypanosomiasis. Leber, therefore, concluded that a toxin, and not an actual invasion of the trypanosome, was responsible for the corneal opacity. To further prove this, Leber infected mice with trypanosomes, and then rendered their blood sterile by intravenous injections of Ehrlich's "Spirasyl." This sterile or "cured" blood, which should supposedly contain the trypanosome toxin, when injected into the anterior chamber of rabbits, caused an exquisite keratitis in six to eight days. This last experiment is invalidated, however, by the observation of Sattler (10), in 1909, that normal foreign blood injected in the anterior chamber can cause similar lesions.

Morax (11) again, in 1909, reported experiments in which he attempted to produce keratitis by intracorneal injection of aqueous and blood containing trypanosomes. Morax observed the precaution of injecting aqueous and blood from animals of the same species, avoiding the lesions incident to the injection of foreign protein. A keratitis or corneal opacity did not occur in any of the animals injected intracorneally.

Morax, therefore, stated that it was impossible to say whether the corneal lesions resulting from trypanosome infection were produced by a toxin elaborated by the trypanosomes in the anterior chamber or by the actual invasion of the cornea by the trypanosomes.

The occurrence of trypanosomes in the tissue of the cornea was substantiated in 1911 by Yorke (12). In excellent studies Yorke conclusively showed the presence of trypanosomes in the areas of exudation in the cornea. To demonstrate the trypanosomes it was necessary that the cornea be fixed in Flemming's solution, and the sections stained either by the iron-alum-hæmatoxylin or by the safranine-methylene-blue-orange-tannin methods. If the tissues were fixed in formalin, the trypanosomes were not stained.

Ulbrich (13), in 1910, studied the chorioretinitis produced by trypanosomes. It was impossible to study such lesions in animals with a general trypanosomiasis because the corneal opacity was of such intensity that it was impossible to see the fundus. Ulbrich isolated the vortical vein of the orbit and injected trypanosomes there. By this means he was able to produce a chorioretinitis in 4 out of 15 animals. He was unable to demonstrate trypanosomes in the tissue of the fundus, but found them constantly in the fluid beneath the retina.

In relation to the formation of toxins by the trypanosome, Laveran and Pettit (14) have conclusively demonstrated that an endotoxin is produced which may give characteristic symptoms.

TECHNIC.

The Trypanosoma equiperdum was used. A dog was inoculated intraperitoneally with the blood of a white rat infected with such trypanosomes. About ten days later trypanosomes appeared in the circulating blood. The dogs used in the experiments were now infected from this or from similar seed dogs. To infect the dogs, 1*cc* per kilo of body weight of the blood of the seed dogs was given intravenously.

GENERAL SYMPTOMS.

No change was noted in the dogs for a period of from one to two weeks. Usually at some time during this period trypanosomes appeared in the circulating blood. The dogs then showed an intense, gradually developing anæmia and loss of weight. As successive dogs were inoculated, the trypanosome infection became more virulent. The dogs became drowsy and listless and slept constantly. Localized swellings due to œdema appeared over the body, in the joints, scrotum, etc., and trypanosomes were found in the œdema fluid. In fact, there appeared to be an invasion of trypanosomes in practically every fluid of the organism. Enlargement of the spleen, choluria, and increased urobilin excretion were constant findings.

OCULAR SYMPTOMS.

Incubation Period.—The interval elapsing between the time of inoculation and the occurrence of trypanosomes in the blood was variable, and depended entirely upon the amount and the trypanosome content of the blood with which the dog was infected. After the appearance of trypanosomes in the blood, the interval elapsing before the occurrence of ocular symptoms was variable. Ocular symptoms appeared in one dog two days after the appearance of trypanosomes in the blood, in another three days, in another five days, and at irregular intervals up to eighteen days. Three dogs died from trypanosomiasis—two on the seventeenth day after the appearance of trypanosomes in the blood and one on the fourth day, without the development of eye lesions. In general, however, in the dogs which developed ocular lesions, the symptoms appeared usually between the fifth and the twelfth days after the appearance of trypanosomes in the blood.

TYPES AND CLINICAL COURSE OF OCULAR LESIONS.

Two distinct types of ocular lesions were noted: (1) A simple clouding of the cornea with little pericorneal injection, photophobia, or involvement of the iris; (2) a clouding of the cornea with marked pericorneal injection, photophobia, iritis, and hemorrhages and exudations in the anterior cham-

ber. In the dogs first inoculated the simple clouding of the cornea alone was observed. As the trypanosome was transferred from dog to dog and became more virulent, the development of the iritis became more constant and severe in character.

In several dogs the first symptom observed was a marked contraction of the pupil, with an elevation of the iris and a shallow anterior chamber, giving the appearance of an "iris bombé" due to a posterior effusion. This was followed in twenty-four hours by a clouding of the cornea. In the other dogs this preliminary contraction of the pupil and shallow anterior chamber were not observed.

In two dogs showing only the clouding of the cornea without iritis, a very slight pericorneal injection first appeared, which was followed in twenty-four hours by a slight dimming of the cornea, which gradually increased in intensity until the entire cornea was gray and opaque and the iris could not be seen. Examined with a sph. + 20 D. lens, the opacity of the cornea was uniform, resembling ground glass. This condition of the cornea persisted for from four to seven days, gradually clearing up, leaving an apparently normal cornea, iris, and fundus.

The majority of the dogs showed an iritis in addition to the keratitis. These dogs developed first a very slight pericorneal injection, followed by a slight haze of the cornea. Synchronous with the development of the opacity of the cornea, hemorrhages appeared in the anterior chamber. The iris was irregular and a slight exudate appeared. The pericorneal injection deepened, the cornea became gray and opaque, the exudate and hemorrhage in the anterior chamber increased in size, and a marked photophobia developed—in short, the symptoms of an exudative iritis with keratitis. This condition continued and became gradually more intense up to the death of the animal. In no case were granuloma of the iris or corneal ulcers observed.

CHARACTER OF THE AQUEOUS AND VITREOUS HUMORS.

In the aqueous of all eyes showing lesions, trypanosomes were found. The aqueous of those eyes with only a simple

keratitis contained trypanosomes and occasional endothelial cells. The aqueous of the eyes with both a keratitis and iritis showed trypanosomes, many red blood-cells, endothelial cells, small round-cells, and leucocytes. Active phagocytosis of the erythrocytes by the leucocytes was going on. The endothelial cells appeared from their morphology to be either from the endothelial layer of the cornea or from the anterior epithelial layer of the iris.

It was impossible to make examinations of the vitreous from the eyes of living dogs. To determine the character of the vitreous an eye showing the typical lesions, in a dog infected with trypanosomes, was enucleated. The eye was opened, and a portion of the vitreous removed. The vitreous appeared much less viscous than normal. Examined microscopically, this vitreous contained a moderate number of red blood-cells and occasional actively motile trypanosomes. No other cells were seen.

We have made repeated examinations of the blood and aqueous, noting the presence or absence of trypanosomes and the time of occurrence of eye lesions. Three dogs of the series ran a typical general course of trypanosomiasis, anæmia, enlarged spleen, etc., without the occurrence of trypanosomes in the aqueous or the development of eye lesions. Every dog with eye lesions, however, showed trypanosomes in the aqueous. Ocular lesions are apparently not due, therefore, to a general toxin carried to the eye by the blood or lymph.

Repeated examinations of the aqueous have been made before, synchronous with, and after, the appearance of the ocular lesions. In no case were trypanosomes found in the aqueous without the development of eye lesions. With the appearance of the first faint symptoms of impending eye trouble trypanosomes constantly appeared in the aqueous. Several times, in the routine examination of the aqueous made every two days, trypanosomes would be found in the aqueous of an eye apparently clear. In each such case the next examination of the eye, always within eighteen hours and usually before, showed beginning ocular disorder. The development of the ocular lesions is synchronous with, if not dependent upon, an infection of the aqueous with trypanosomes.

In order to demonstrate this point further, the anterior chamber of one eye of a normal dog was injected with the trypanosome-containing aqueous of a dog with ocular lesions and a general trypanosomiasis. The next day this dog showed a contracted pupil and a shallow anterior chamber, with a clear cornea—the earliest symptom of impending ocular disorder due to trypanosome infection. Twenty-four hours later there was a well-marked opacity of the cornea, with pericorneal injection, which was soon complicated by an iritis. The blood of this dog remained free of trypanosomes for some time, no anæmia developed, and beyond the ocular condition the dog remained normal in every way.

In relation to the hemorrhages observed in the anterior chamber, thrombosis of the capillaries, especially of the brain, has been observed several times by other authors. Although we have no direct proof, it seems quite probable that the hemorrhages in the anterior chamber may be caused by a thrombosis or rupture of the arteries of the iris by the trypanosomes.

POSSIBLE RELATIONSHIP OF OTHER CONDITIONS TO OCULAR LESIONS.

All dogs injected with trypanosomes showed a decided anæmia and enlarged spleens. As will be shown in other studies from this department, the anæmia was due to increased blood destruction, which was only partially compensated for. This was demonstrated by the choluria and the increased urobilin excretion. It seemed possible that these factors might have some influence on the ocular lesions. In order to determine this, the blood-picture of dogs developing eye lesions and those without eye lesions have been followed. No appreciable difference was found between the two, and it seems probable that the anæmia bears no relationship to the eye lesions. Tables I. and II. illustrate this point:

TABLE I.—ANÆMIA OF DOGS SHOWING OCULAR SYMPTOMS.

Dog.	Control at Time of Inoculation.			At Time of Appearance of Trypanosomes in Blood.			At Time of Outbreak of Ocular Symptoms.		
	Hb, per cent.	R. B. C.	W. B. C.	Hb, per cent.	R. B. C.	W. B. C.	Hb, per cent.	R. B. C.	W. B. C.
16–69...	96	7,210,000	15,800	72	6,006,000	12,600	31	3,600,000	5,000
16–72...	101	6,400,000	10,200	46	2,800,000	10,660	30	2,900,000	25,600
16–76...	101	7,300,000	9,000	60	4,080,000	24,000	61	3,660,000	15,200
16–78...	103	6,580,000	12,400	90	6,600,000	11,600	50	3,400,000	5,400
16–93...	116	7,450,000	10,800	114	7,800,000	9,000	84	4,720,000	16,130
16–94...	112	7,500,000	11,200	68	4,500,000	10,500	62	3,870,000	9,900
16–97...	112	7,430,000	22,700	96	7,600,000	17,000	32	2,700,000	4,500
17–23...	98	7,240,000	14,000	99	6,410,000	12,500	65	4,310,000	5,200
17–30...	90	6,300,000	16,000	76	5,200,000	11,200	52	3,660,000	6,600

TABLE II.—ANÆMIA OF DOGS WITHOUT OCULAR LESIONS.

Dog.	Control at Time of Inoculation.			At Time of Appearance of Trypanosomes in Blood.			Fifteen Days after Appearance of Trypanosomes in Blood.		
	Hb, per cent.	R. B. C.	W. B. C.	Hb, per cent.	R. B. C.	W. B. C.	Hb, per cent.	R. B. C.	W. B. C.
16–79...	83	5,000,000	12,000	62	4,200,000	8,400	39	3,000,000	5,600
17–44..	111	7,018,000	13,000	80	5,660,000	12,100	44	3,970,000	5,700

Similarly, several dogs with trypanosomiasis and eye lesions have been splenectomized to determine if splenectomy could have any influence upon the disease, and consequently upon the ocular condition. The dogs splenectomized did not do well, the general condition and the ocular condition remaining practically unaffected, or, if anything, becoming more intense. All the dogs splenectomized died within ten days, with no improvement in the ocular condition.

MICROSCOPIC PATHOLOGY.[1]

Lesions are present in the cornea, iris, and retina. The lesions shown in the cornea of those eyes showing only a simple keratitis, and those with both a keratitis and an iritis,

[1] We are indebted to Dr. Allen J. Smith, Professor of Pathology in the University of Pennsylvania, for valuable assistance in the study of the Sections.

are similar in character, differing only in intensity. The lesions are best shown by hæmatoxylin and eosin staining. The lesions caused by trypanosomes in the several tissues of the eye are as follows:

CORNEA.—*Hæmatoxylin and Eosin Stain.*—The cornea is two or three times its normal thickness. The lesions are limited entirely to the substantia propria. This tissue is very loose and distended; some foci are much looser than others. The distention of the cornea is intralamellar. Invading these foci, and throughout the cornea, there is considerable cellular infiltration, in which polynuclear and mononuclear cells can clearly be recognized. In addition there are elongated forms which, from their granularity, are probably polynuclear cells, while other elongated cells with more solid nuclei are probably fibroblasts. Elongated spiral-shaped cells are also present which have the external morphology of trypanosomes, but in the corneal sections, with this stain, the characteristic nuclear arrangement, upon which a definite diagnosis can be made, cannot be demonstrated.

There are new capillaries extending up from the sclera into the cornea. These capillaries are choked with blood (Fig. 1).

Alum-Iron-Hæmatoxylin Stain.—Yorke (12) has recommended for the demonstration of trypanosomes in the cornea that the tissue be fixed in Flemming's solution, embedded in paraffin, sectioned as thin as possible, and stained by alum-iron-hæmatoxylin (Heidenhain's method). Preparations of a cornea showing the characteristic lesions have been made and stained by this method.

In these sections, especially when counterstained, trypanosomes can be definitely made out in between the lamellæ of the cornea. In the sections we have prepared the parasites are not as numerous as in the sections reported by Morax and Yorke. Nevertheless, they can be made out, and the characteristic nuclear arrangement demonstrated.[1] The protoplasm stains very poorly with the counterstain (Fig. 1, B).

[1] These sections have been submitted to two pathologists, Dr. Allen J. Smith and Dr. Richard M. Pearce, who have both concurred in the definite diagnosis of trypanosomes in the cornea.

Romanowsky (Wright's) Stain.—In the hope of rendering the trypanosomes more distinct, a number of sections have been stained by Romanowsky's method. With this stain the nuclei stain a dark red or purple, while the protoplasm stains blue. Trypanosomes in the cornea, lying in the substantia propria, can be demonstrated by this means.

Giemsa Stain.—A number of sections have been stained by the Giemsa method, in the hope of demonstrating trypanosomes. These preparations have been unsatisfactory. While the same suggestive bodies seen with hæmatoxylin and eosin staining are again seen in the cornea, the characteristic nuclear arrangement cannot be seen, and a definite diagnosis of trypanosomes in the cornea cannot be made by this stain.

Levaditi's Silver Nitrate Stain.—Sections from a number of eyes have been stained by this method. The same difficulties met with in the other stains are again encountered here, and the definite presence of trypanosomes in the cornea cannot be established by this means.

Iris.—There is considerable distention and looseness of the stroma of the iris. In these areas of distention there are a good deal of granular material, a moderate amount of fibrin, a large number of small round cells, greatly necrosed, and a number of polynuclear cells. There is no evidence of trypanosomes in this tissue with any of the several stains used. Over the anterior epithelial layer there is a thin exudate of a gelatinous, homogeneous material containing cellular elements.

The ciliary body and ciliary processes themselves appear to be practically unaffected. In a few eyes the stroma of the ciliary processes shows a very slight cellular infiltration, similar to that present in the stroma of the iris. The pigment layer always appears to be normal. However, there is a very definite exudate around the ciliary processes, of a similar character to that found over the iris. The exudate is serofibrinous in character, and is densely infiltrated with mononuclear and polynuclear leucocytes. In this exudate a few trypanosomes can be seen. (Fig. 2.)

Retina.—The changes in the retina consist of small focal lesions, varying greatly in size. These lesions are manifested by a slight thickening of the retina, a general looseness of the structure, indistinctness of the ganglion-cells, fullness of the

capillaries of the vascular layer, and a general diffuse round-cell appearance of the deeper layers, including the rods and cones. Much of this seems to be of a monocellular type, which may have come from the diffusion of the cells of the nuclear and granular layers. Polynuclear cells cannot be distinguished, but they may be present and obscured by the lymphocytic infiltration. Over the surface of these foci is a very faint eosin-staining homogeneous material, in which are polynuclear cells and a number of badly vacuolated rounded cells, containing one or two nuclei. These are probably mononuclear cells of the retina proper in a degenerate condition. In this surface exudate there are also a number of globules, homogeneous and hyaline in appearance, faintly staining with eosin. Here and there in between these cells degenerate and rather poorly stained trypanosomes are easily and definitely recognized. These trypanosomes were not recognized in the retina proper. (Fig. 3.)

In other portions of the fundus, between the pigment layer of the choroid and the sclera, there are large, serofibrinous cellular exudates containing chiefly degenerated mononuclear cells and red blood-cells.

THERAPY.

Riquier (15), Schamberg, Kolmer, and Raiziss (16) have shown that salvarsan, and the identical American production, arsenobenzol, have a chemotherapeutic effect in trypanosomiasis. In order to determine if a similar therapeutic effect is exerted upon the ocular condition, two dogs with a general trypanosomiasis and ocular lesions and one dog inoculated in the aqueous (ocular lesions without a general trypanosomiasis) have been treated intravenously with arsenobenzol. Two dogs were given three injections—0.10*gm* per kilo of body weight at each injection, on successive days. Three days after the last injection, the blood and aqueous of one dog were free of trypanosomes and the eyes were clearing rapidly. Within one week the ocular lesions had completely disappeared and the eyes appeared normal. Similarly the diseased eye of the dog inoculated in the anterior chamber rapidly cleared, the aqueous being free of

trypanosomes on the day of the last injection, and the eye lesions becoming entirely healed within ten days.

One dog was given only a single injection. The trypanosomes disappeared from both the blood and aqueous within two days, the eyes becoming entirely clear within eight days. Following the arsenobenzol injection the dog appeared much livelier and the blood-picture slowly improved. Nineteen days later, however, trypanosomes again occurred in the blood, an anæmia appeared, and nine days later ocular symptoms returned. One injection of arsenobenzol was evidently not sufficient to effect a permanent cure.

COMMENT.

These lesions—keratitis, iritis, and retinitis—caused by the trypanosome bear a most striking similarity to those caused in man by the Treponema pallida. In these lesions, as in those of syphilis, there is an actual invasion of the tissues by the parasite. The corneal lesions caused by the trypanosome and by the spirochæte appear pathologically to be the same intrinsic process—an interstitial keratitis.

These lesions are manifestly due: (1) To a general toxin carried either in the blood or lymph; (2) to a toxin elaborated directly in the eye by the trypanosomes; or (3) to an actual invasion of the ocular tissues by the trypanosomes. The first possibility, that the lesions are due to a general toxin, can be definitely eliminated, for the ocular lesions do not occur in an animal, no matter how heavily infected, independent of an actual invasion of the ocular humors by the parasites. Whether or not the corneal lesions are due to a toxin elaborated in the humors, or by an actual invasion of the trypanosomes in the substantia propria, cannot be said. The experiments of Morax (11) on the intracorneal injection of trypanosomes are inconclusive. In every case with us the development of corneal symptoms was synchronous with the appearance of trypanosomes in the aqueous humor. Although trypanosomes were found in the cornea, we cannot say whether or not they appeared before or after the development of the keratitis. Trypanosomes were not found in the lesions of the iris, but this failure may easily be due to the

thicker structure and pigment of the iris obscuring any parasites that may have been present. Trypanosomes were found in the exudate over the lesions in the retina, but not in the retina lesions. Again, this may well be due to greater density of the retinal tissue and the dense cellular infiltration. To us the most logical view appears to be that both factors—the actual invasion of the tissues by trypanosomes and a toxin elaborated by the trypanosomes in the humors—play a part in the development of the ocular lesions.

The action of the arsenobenzol upon the lesions is of especial interest. The subsidence of all symptoms and the return of the eye to an apparently normal condition are so prompt that there must be a very prompt excretion of arsenobenzol into the ocular humors. If the corneal lesions are due to a toxin elaborated by the trypanosomes in the substantia propria of the cornea, there must be an equally prompt excretion of the arsenobenzol by the lymphatics of the cornea. In either case it is of interest that corneal lesions, similar to those of an interstitial keratitis of syphilitic origin, respond so promptly and readily to arsenobenzol.

SUMMARY.

Ocular lesions occur constantly in dogs infected with the Trypanosoma equiperdum. These lesions are manifested clinically as a dense clouding of the cornea and an iritis with hemorrhages and exudates in the anterior chamber, with the occurrence of pericorneal injection and photophobia. The lesions are not caused by a general toxæmia, but occur synchronously with an actual invasion of the ocular humors by the trypanosome, and appear to be dependent upon the invasion of the ocular humors and the ocular tissues by the parasites. The ocular lesions are readily cured by repeated injections of arsenobenzol, the American reproduction of salvarsan.

Pathologically, the corneal lesions are those of an interstitial keratitis with an actual invasion of the substantia propria by the trypanosomes. The lesions of the iris consist in a marked œdema of that tissue, with a rather dense cellular infiltration of mononuclear and polynuclear cells. The ciliary

processes are themselves uninvolved, but occasionally they are embedded in a serofibrinous cellular exudate in which occasional trypanosomes are found. The retinal lesions consist of focal lesions characterized by a disarrangement of the retinal layers with a monocellular infiltration. Over these areas is a cellular exudate containing trypanosomes.

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THE TONSIL AS A SOURCE OF INFECTION IN IRITIS AND FOCAL CHOROIDITIS.

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THE especial objects of this paper are to comment upon the tonsillar crypts as foci of infection for certain types of intraocular inflammation, and upon the value of vaccine therapy in their treatment. Inflammation of the iris will first be considered.

I think it may be taken for granted that all forms of iritis, save the traumatic, are infectious in origin. Excluding the tubercular, the venereal, and certain rare forms, *e.g.* the leprous, the focus of infection will most frequently be found in the mucous membrane of the upper air tract. This includes also the sinuses, the ear, and the dental region. With the exception of the peridental tissues, it is highly probable that a chronically infected tonsillar crypt is more frequently the origin of inflammation of the iris than disturbances anywhere else along the upper mucous membranes.

Were it not for the crypts, those mucous-lined recesses within the tonsils, tonsillar inflammation and hypertrophy would be among the rare diseases. In the beginning the production of tonsillitis is the result of mechanical conditions springing from the form and location of the crypts. Primary inflammation of the mucous membrane covering the lining of the inner surfaces of the cheeks is extremely rare. Tonsillitis is a very common disease. Why is this so? The same germs are present from time to time on the surfaces of the mucosa of the latter as are to be found on the surface of the tonsils. The explanation is to be found in the fact that the inner surface of the cheeks has no recesses in which the germs of the

mouth may accumulate and multiply. The crypts of the tonsils on the other hand, furnishing, as they do, heat, nourishing food, and rest, are ideal places for germ growth. In early life the continued presence of micro-organisms and their products within the crypts results in tonsillar hypertrophy. As years go by, for reasons out of place here, this hypertrophy to a greater or less extent disappears and it frequently happens that the crypts are left enlarged and so distorted that their contents cannot be emptied during the act of swallowing. *The continued presence within the crypts of the cast-off epithelial cells*, live and dead micro-organisms and their products, results in the formation of the well-known dirty, whitish, stinking masses and also, sooner or later, as the result of pressure, in the destruction of the epithelial lining of the crypts, and often in superficial ulceration of the underlying tissues. These three things may persist in varying degrees throughout life and the passage into the blood of the germs and their products present in the crypts is often the cause of inflammation in distant organs resulting in, *e.g.*, thyroid hypertrophy, articular rheumatism, appendicitis, endocarditis, etc. It seems to have been proved that different strains of the same organism select different parts of the body for their action. In this connection it must be borne in mind that where the cryptal mucous membrane becomes ulcerated, and it often does, there results a more or less constant passage into the blood of micro-organisms and their products.

In examining the tonsil the anterior faucial pillar should be pulled aside and a blunt probe passed into each crypt. In especial should the uppermost one be examined. Should we find present the above mentioned whitish masses we may be fairly sure that the mucous membrane lining the crypt is diseased, that either its covering epithelium is absent in spots or that the lining is ulcerated. In either case we have a chronic focus of infection. Our examination, however, does not stop here. With the pillar held aside, the tonsil should be pressed upon. At times a gruel-like purulent substance can be expressed from the crypts. Again we have an active focus of infection. Should neither the whitish masses nor the gruel-like substance be present, we should then pass our probe into each crypt and smell the probe thus inserted. Should a foul

smell be found we may be sure we have an actively infected crypt. Lastly we may make cultures from the crypts. When, however, none of these above-mentioned conditions is present, it is less likely, though possible, that the tonsil is a focus of infection. These remarks apply to the chronic forms of tonsillar disease. In acute tonsillitis the infectious focus is obvious. Study of patients who have chronically diseased conditions of the tonsillar crypts has led me to the belief that in all cases there is taking place through an abraded mucosa a slow passage into the blood of a varying amount of micro-organisms and their products. Clinical observations of such patients have revealed a host of symptoms both inflammatory and "nervous," which removal of the tonsils proves to have had their focus of origin within the crypts. So far as the eye is concerned these symptoms are manifested within both the inflammatory and "nervous" domain. If this tonsillar picture be closely borne in mind, it will help us to understand why in the presence of an unremoved focus of infection our "remedies" are of so little value, and why after removal of the focus "remedies" are so often unnecessary. ***It helps us also to understand the recurrence of local secondary inflammations such as iritis, focal choroiditis, scleritis, etc.*** Of the micro-organisms which pass into the blood from the infected crypts the vast majority are destroyed by the blood cells before they reach tissues where the conditions are such that they can intrench themselves and produce local inflammation. When, however, they have once found such a receptive focus and have "inflamed" it, they often leave this focus, even though driven out by "treatment," in such a weakened condition that the same species of micro-organism can later reproduce there an inflammation similar to the first. Consideration of the pictures presented in chronic tonsillar crypts will make it clear how in the peridental infection we have to deal with similar conditions. Of the two sources of infection, the tonsillar crypts and the peridental fissures, the latter when once infected furnish anatomical conditions more favorable for absorption of the infection material than the crypts, and are less amenable to local treatment other than removal of the tooth. It often happens in a case of iritis or focal choroiditis, for example, that we find disease of both the dental regions and the tonsillar

crypts, and it is impossible to decide from which of the two the micro-organic invasion which has resulted in the ocular inflammation is taking place. It will be necessary in such cases to remove both foci to obtain the cure we are seeking.

In considering the question whether a permanent cure of the inflammations under consideration will result from the complete removal of the infected focus whether in a tonsil or about a tooth, we must bear in mind that the removal, while it may do away with the cause and result in a rapid cure of the inflammatory condition we are treating at the time, we have by no means done away with the various species of the coccideæ resident along the mucosa of the upper air tract, and that recurrences may occur whenever at any spot such a condition of the mucosa arises that will permit the passage of these micro-organisms through it into the blood. Of especial interest in this connection is the intranasal membranes, the relation of whose mucosa, when diseased, to intraocular inflammation furnishes a most interesting and instructive series of lessons. None perhaps more so than that type of optic atrophy which we find so often accompanying the fetid, dry catarrh of old people. This is mentioned here, although apparently out of place, because it brings out the question as to whether the toxines absorbed from a chronically infected focus can produce distant local trouble as well as the absorption of the germs themselves. I believe they can and do and that time will unfold a distinct demarcation between the two sets of pathological manifestations. In the treatment of intraocular secondary inflammations the condition of the gastro-intestinal tract must never be lost sight of. The conditions present in this region in what we term a "common cold" unquestionably lessen the germ-destroying properties of the blood. "Common colds" are of two kinds. One begins with a congestion of the digestive mucosa; the other with an inflammation of the nasal mucosa. The manifestations of the former in the nose are varied by the resulting concurrent activities of the micro-organisms previously present in the nose. The latter is a purely infectious disease, and the treatment of the two varieties should accordingly be different. I have called attention to this because of its extreme importance in the treatment of all acute secondary intraocular local

inflammations, and, secondarily, because in the cases under consideration the condition of the digestive tract influences the effect of the vaccine upon the ocular trouble.

Concerning the use of vaccines in the treatment of iritis. Everyone who has had any, even a very limited, experience with the use of vaccines, whether autogenous or stock, for acute infectious inflammations must have been struck with two facts. One, the rapid amelioration of the inflammation following the use of the vaccine in certain cases. The other, the apparent failure of the vaccine to influence the inflammatory process in other cases. From the theoretical standpoint the autogenous vaccines seem clearly to have an indisputable superiority. On the practical side some of the stock preparations have claims which cannot be denied. In cases of iritis, choroiditis, scleritis, episcleritis of the secondary type, it is probably true, as stated above, that in the vast majority of the cases the focus of infections will be found in the mucous membrane of the upper air tract. This being so, the most common micro-organisms we have to deal with belong to the family of the coccideæ, a peculiarity of which is that, up to the present time, the science of medicine has not been able to find a vaccine which can procure for the members of the human family more than a very limited immunity from their activity. In secondary intraocular focal inflammations it is often from a practical standpoint impossible to certainly determine the especial coccus causing the inflammation. In the diseased tonsil crypts, and the same is true for the diseased areas about the roots of the teeth, bacteriological examinations, as a rule, reveal the presence of several micro-organisms, and the question of the autogenous vaccine proper for a given case is not always easy to decide. Again, the preparation of an autogenous vaccine requires time, and time is very important in the treatment of intraocular inflammations. More than eighteen months ago I began to experiment with the use of Mulford's mixed influenza sero-bacterins. Results obtained in certain acute infectious inflammations of the upper air tract led me to try them in iritis. The results of these experiments are worthy of report. The preparation just mentioned contains "killed sensitized bacteria as follows: B. influenzæ, staphylococcus albus et aureus, streptococcus, pneumococcus, the

catarrhal group." The local reaction following its use is slight and a general reaction relatively rarely to be observed. The results in the first two cases in which I made use of this preparation were so striking that I have since then used it in all cases of iritis of the type under discussion. In no case have I, however, failed to supplement its use with such general local treatment as I thought the case to require. That in the large majority of cases it rapidly and favorably modifies the acute inflammatory process is beyond question. Its use in most cases is followed by a relatively rapid disappearance of the exudate from the iris. It is a valuable adjunct to our list of remedies in the treatment of ocular secondary inflammations. It cannot be relied upon when used by itself to bring about a permanent cure. Some of the reasons for this are commented upon at length in the discussion of the cases herewith reported. Its immediate effects are often sufficient to result in the destruction of the active germ life at the seat of the iritic inflammation. I have not convinced myself that it in any way produces changes in the infected primary focus in the tonsillar crypts or about a tooth. It assists the blood in destroying the infectious agents on their way from the infected focus to the iris. This assistance, however, is temporary and unless the primary focus be removed the infectious agents will again find their way into the blood in sufficient quantities to cause a recurrence. Though this be true, I know of nothing which will, in the majority of cases, so rapidly clear up the region of inflammation in cases of iritis, provided it be used in the earlier stages of the trouble and when there are not present concurrent marked evidences of intestinal disturbance as shown by a furred tongue and foul breath; or where there is not reaching the iris an overwhelming amount of the infection. For example, in gonorrheal iritis, while the use of the Neisser sero-bacterin, in my experience confined to two well-marked cases, occurring in the gleet stage, produced a marked amelioration of the iritis inflammation within two days, not less than eight injections were required to obtain a disappearance of the active inflammatory symptoms, and in one of these cases the injections had to be repeated for a recurrence within three months.

CASE REPORTS.

On September, the 20th, 1915, A., aged 28, consulted me in regard to a right-sided recurrent iritis. The attack had lasted several weeks. The patient was tubercular in appearance, with poorly developed arms and legs, the skin of which seemed lacking in supporting tissue. To look at him one would say any inflammation he might have would be difficult to treat. The iritis had been present several days when I saw him. In spite of internal treatment and the frequent use of atropine continued for ten days, the pupil, although dilating regularly, refused to dilate more than ⅓ of the amount it should. The eye grew steadily worse and many fine deposits appeared in Descemet's membrane. Apparently treatment was having little effect on the severity of the inflammation. In this case I noted no focal infection of the upper respiratory mucosa, only a tendency to a general increased secretion such as is present in all cases of active pulmonary tubercular infection. It is possible that in both this case and the following one there may have been some mild tonsillar infection, as I was at that time not as fixed in my ideas as to tonsillar infection being so often the cause of iritis as I now am. At a venture I gave him the first dose of Mulford's influenza sero-bacterin mixed. The following day the appearance of the eye had entirely changed. The pain, the photophobia, the symptoms of acute inflammation had disappeared. In their place the eye had the appearance of being far on the road to recovery after having been the seat of severe inflammation. There was no return of the inflammation although some little while was required for the disappearance of the visible exudates in Descemet's membrane. In this case three more doses of the serobacterin were administered at intervals of one and two days.

On Oct. 12, 1915, M., aged 48, consulted me about her right eye, which had been the seat of an iritis for eight weeks, during which time she had been under constant treatment which, while lessening, had not been able to remove, the symptoms of acute inflammation. While dilatation of the pupil had been secured, there were visible three patches of exudate around the edge of the pupil and the details of the fundus were imperfectly to be seen. The teeth were normal and I noted no antral or tonsillar disease. On Oct. 13th, the first dose of Mulford's influenza sero-bacterin mixed was given. On the 14th, the patient reported that she had slept well during the previous night and that the pain was not so severe. The acuteness of the inflammation was visibly less. The second dose of the mixed bacterin was

given on the 15th. A third dose was administered on the 16th. On the 18th, the eye was no longer inflamed; the exudates in the anterior chamber, cornea, and lens had so far cleared up that the details of the fundus were fully visible. The eye healed at once without anything of further note.

In October, 1916, A. J., aged 27, came to see me about his right eye. He was a tuberculous-looking man, whose left eye was partially atrophied as the result of a blow received some years before. The right eye was the seat of an acute iritis of eight days' standing. Examination of the nose, throat, and teeth revealed no focus of infection. As the patient was unable to remain in Richmond for treatment, I ordered atropia and the usual general treatment. Three or four days later he returned with all the usual evidences of an iritis increasing in severity. Syringe A of Mulford's influenza sero-bacterin mixed was injected. The following day, no improvement being visible, Syringe B was injected. The next morning the pain was gone and there had appeared that whitish appearance of the episcleral tissues which indicates the disappearance of inflammatory activity. Syringes C and D were injected the two following days and the eye was well. Here was another case of iritis, which under the usual and general treatment would, I feel sure, judging from years of experience, have required two weeks or longer at the least to have been cured.

These three cases sufficiently illustrate the value of the influenza sero-bacterin mixed, where we can discover no focal cause for the inflammation and where the obtainable history tells me of a "cold in the head" and a concurrent digestive upset. In these cases, I am inclined to think the organisms causing the iritis passed into the blood current from some slight abrasion of the upper mucosa during the cold. This, however, may be incorrect.

The cases of A. and M. were the first in which I used the mixed sero-bacterins and the results obtained made me more than willing to continue their use in other cases. I was to find, however, that they have very decided limitations as therapeutic agents. In neither of these cases did I discover a focus of infection. In both of them I left it unremoved, and in both cases more than a possibility of a future recurrence of the iritis exists. As case reports the above three cases are valuable

only as showing the possible results following the use of the mixed sero-bacterins. They teach nothing else. My experience with later cases led me to the belief that, where by the third or fourth day after the first injection there was not a very marked diminution of the inflammatory symptoms, the amount of infectious material reaching the iris was so great that no dependence could be put upon the sero-bacterins to bring about a cure unless the primary focus should have been removed. The injections are given as follows: First day, Syringe A; second day, Syringe B; fourth and sixth days, Syringes C and D.

J. K., aged 26, seen first Jan. 28, 1917. Right eye seat of iritis of five days' standing, apparently of that mild type which usually responds readily to remedies. Patient has a brother who has had eight attacks of iritis and a sister who has had five. Five years before, he had had rheumatism, which caused severe pericarditis, and, attacking his feet, caused extreme bilateral flat foot so that despite operative measures he has to use crutches. No history of gonorrhea. Examination of the throat shows a left tonsil from which exuded a gruel-like pus. The right tonsil had within its crypts whitish masses. Owing to the condition of the heart it was decided to treat the tonsil conditions locally. In spite, however, of daily use of the mixed sero-bacterins, and of active general and local treatment and careful attention to the tonsillar trouble for three days the intensity of the iritis increased. On Feb. 1st the left tonsil was removed. The crypts of the right tonsil were by this time apparently clean. By Feb. 4th, the inflammation had greatly involved the uveal tract and the vision had become perception of light. On Feb. 5th, Syringe A was administered. On Feb. 6th, there could be seen over the outer part of the anterior surface of the iris a small hemorrhage. On Feb. 7th, following a dose of sero-bacterins mixed on the 6th, the eye became more comfortable and there appeared in the distant parts of the sclera that whitish color which denotes the subsidence of the active inflammatory stage. Two more doses of the sero-bacterin were administered, one on the 8th, and one on the 9th, when the patient returned home with eye practically well. This case is instructive. Apparently at the beginning it was one favorable for the use of the mixed bacterins and yet their administration seemed in no way to check the inflammatory process. I think it is almost certain that the source of iritic trouble was the left tonsil,

which although but slightly hypertrophied had, when I first saw it, a thin muco-purulent fluid exuding from its crypts. It is highly probable that the strain of the organism in the crypts was one to which case J. K. was unduly susceptible, and further that there were abrasions in the mucous linings of the crypts through which the micro-organisms were pouring into the blood and so reaching the iritic region. The sero-bacterins were ineffective probably because the local efforts to destroy the germs within the tonsil crypts were a failure and possibly resulted in the causation of further lesions in the mucous lining of the crypts, which rather increased than decreased the ease with which the micro-organisms entered into the blood. As soon, however, as the tonsil was removed the sero-bacterins then administered became effectual and the inflammation of the iris rapidly disappeared. The right tonsil was removed after the subsidence of the iritis. Had it not been for the condition of this patient's heart both tonsils would have been removed at once. After the removal of the left tonsil, whatever micro-organisms were entering the circulation from the right one were destroyed by the blood, which with the aid of the sero-bacterins allowed of a rapid subsidence of an inflammation which at one time seriously threatened the vision. Had both tonsils been removed at the beginning it is not unlikely that the iritis would have subsided far more rapidly than it did. The practical points of all this for ophthalmologists are several. (1) The patient's word can never be accepted as to the tonsillar condition. I have seen a patient of forty-two who "never had a sore throat in her life" and yet all of whose tonsillar crypts were filled with semi-solid muco-purulent masses. She was a cripple from rheumatism which disappeared after the removal of the tonsils. (2) A superficial examination of the tonsillar region is worthless. The size of the tonsil is no criterion of its condition, as a source of infection. Many of the most active foci of infection are found in very small tonsils which are to be seen at all only after the anterior pillars of the fauces have been pulled aside. (3) The absence of pain or discomfort in the tonsillar region is no sign whatever that the tonsils are healthy. (4) It is highly probable that infection to a varying extent is constantly taking place from all crypts which contain the well-known "cheesy masses." I believe that in all of such crypts the epithelium would, under microscopic examination, be found to a greater or less extent absent as the result either of pressure from the cheesy masses or of ulceration. (5) In all cases of iritis where the cause is not evident, the tonsils should be carefully examined. (6)

The fact that the tonsillar crypts are found to contain "cheesy masses" or a "gruel-like exudate" does not necessarily mean that this is the only source of the iritic infection. Another focus elsewhere may exist. (7) It is not unlikely that when chronic tonsillar infection is the source of iritic infection there will be recurrent attacks of iritis at various intervals as long as the tonsillar focus remains unremoved. (*Vide* the case of A. P.) (8) In my experience, treatment other than removal of a chronically infected tonsil has proved to be a failure. If we consider the anatomy of the crypts and the condition of the tonsillar substance this can be readily understood.

L., aged 42. Seen first March 24th. Left eye had been painful for ten days. Examination revealed a mild type of iritis. The pupil dilated well under atropia, but the usual triangular area of spots in Descemet's membrane was visible. Examination revealed the fact that both tonsils had in one or more crypts a muco-purulent substance. The second molar tooth in the left side was decayed and painful. The tonsillar crypts were washed out with a boric solution and a mixture of equal parts of tincture of iodine and benzine was added to their surfaces. In addition, Syringe A of Mulford's mixed sero-bacterins was administered. The same treatment was administered on March 25th. On the 26th, all redness of the eye and all the pain had disappeared. On the 28th, Syringe C was administered. On the 29th, the weather turned raw and cold. The patient exposed herself and "took cold." The eye became painful and a slight congestion reappeared. Syringe D was administered and in addition blue mass and salol, *aa* gr. v., were ordered to be taken at bedtime and were to be followed the next morning by a dose of salts. The second day thereafter, the eye was again free from congestion and pain. There was no further trouble until April 8th, when the eye again became congested. The decayed tooth was then extracted and the ocular inflammation permanently and at once disappeared. This case was incorrectly treated. It is reported at length because it offers clearly for comment several points to be borne in mind in the treatment of the types of iritis under consideration in this paper. In the first place the decayed tooth should have been removed as soon as discovered. The difficulties in the way of a certain quick permanent cure of peridental purulent disease are too many to admit of local treatment when we have reason to suspect that this focus is the cause of a local intraocular inflammation. At present I think this admits of no dispute. In the second place the tonsils should have been removed at

once. If we bear in mind the pathological condition of the tonsil, we see at a glance that local treatment of a diseased tonsil must be likewise too uncertain in its immediate results to be given a trial, unless there are contra-indications to its removal, when so important an organ as the eye is the seat of the secondary inflammation. In the third place, the slight ocular congestion and the rapid, although temporary disappearance of this congestion after the use of the sero-bacterins, shows that only a small amount of the infectious agent was reaching the iritic organ. The blood was able to dispose of the greater part of the absorbed infectious agents. The effect of taking cold in the inflamed eye shows that this process lessened the germ-destroying power of the blood and allowed a fresh supply to reach the iris. The importance of the part the condition of the digestive tract plays in the recurrences of local inflammatory conditions should never be overlooked. The disappearance of the iritis after the removal of the tooth shows that the greater part of the absorption took place from the dental region. What about the future of the eye when we recall that the tonsils were not removed? For the present the blood is able to take care of what passes into it from the diseased tonsillar crypts. Whenever, however, the absorption from these crypts is greater than the blood can combat, it is probable that there will be a recurrence of the iritis.

A. P., aged 58. Right eye has an old absolute glaucoma, the result of iritis which had recurred at intervals for several years. The pupil is contracted and entirely adherent to the lens. The left eye has been the seat of a number of attacks of iritis within the past three years. The pupil is contracted and, save for a pinhead area above, entirely adherent to the lens substance. There is a layer of fibrin over the pupil. Patient had received, during the former attacks of iritis, the usual treatment where no definite cause can be found. Patient seen first late in December, 1916. She then had an attack of iritis in the left eye of two or three days' standing. The ocular tension was + 1. The right eye, I inferred, had had no iridectomy performed because of the seeming probability that the exudate had pasted the whole posterior surface of the iris to the lens and for the same reason the left eye had been allowed to pass through several attacks of iritis without an attempt at an iridectomy being made. Examination of the throat revealed the fact that the tonsils had been in childhood the seat of hypertrophy, and while almost all of the hypertrophy had disappeared, the remains thereof showed in the right one a cold abscess half the size of a garden pea. On the

left side one of the crypts contained a cheesy mass. The patient was not conscious of any throat trouble. Besides treatment to the tonsils and the use of atropine, she received three doses of the influenza sero-bacterin mixed, with the result that one week later the attack of iritis had passed off, and the tension had returned to normal. The rapid checking and absorption of the exudate which threatened the destruction of the eye by glaucoma, I attribute to the use of sero-bacterin. (Compare the case of M. as showing the rapid disappearance of a visible exudate within twenty-four hours.) About two weeks later an attempt at an iridectomy was made. The iris was found to be adherent to the lens nowhere except for a band 1½*mm* wide surrounding the pupil. I was unable to pull the iris loose from its pupillary attachment, but I was able to do a satisfactory peripheral iridectomy which showed no tendency to close. The lens was entirely transparent. The case is interesting, not only as showing the value of the sero-bacterin, without whose aid I feel the left eye would have suffered the fate of the right, but because of the tonsillar picture. In this case no-one of her oculists, so far as I could learn from A. P., had ever suspected the tonsils as the constant focus of infection, although she had been subjected to elaborate general examinations with a view to locating the cause of the iritis. This phase of the case is commented on at length in connection with the case of J. K.

H. C., aged 46, seen January, 1917. Large patch of choroidal inflammation in the upper-inner quadrant of the fundus. The quantities of fine opacities in the vitreous tell of an active process. Trouble of some months' standing. Casual examination of the throat reveals an apparently healthy pharynx. When, however, the left anterior pillar is drawn aside, the upper tonsillar crypt is found to be dilated and full of a stinking, whitish cheesy substance. The tonsil was removed. Sections were made to show the condition of the epithelial lining of the wall of the crypt. The epithelial covering at entrance to crypt was normal, while along the walls of the crypt various degrees of ulceration were present.[1] The eye was examined two months later. Apparently the active process in the choroid had subsided. The vitreous, however, contained many floating bodies which had taken the place of the previous dust-like opacities.

[1] Dr. J. G. Dwyer of New York was kind enough to examine this slide and reports that ulceration like this is quite usual in tonsils. He agrees with the writer's conclusions but does not think that this can be shown in the specimen.

I cannot say that I have noted any immediately beneficial effect upon these patches of choroidal inflammation following the use of the sero-bacterins. So far as I know, changes in the vitreous are not susceptible to rapid amelioration following the use of any remedy. The reason for this is, as stated above, to be sought in the structure of the vitreous.

P., age 26. Complained of a "spot before his right eye." It had first appeared about a year previously and had disappeared after several weeks of treatment, to return about two weeks before he consulted me. Examination of his eye revealed in the fundus an area of disturbed choroidal pigment about 3*mm* above the disk. The vitreous contained multitudes of fine spots. No probable focus of infection could be found outside of the tonsils, which "had been removed." Examination of them showed that they had been only partially removed and that the crypts in the remaining portions were infected. Removal of the remainder of the tonsils was followed by improvement in both the patient's general health, and the condition of the eye. I have never seen in these cases that rapid change in the inflammatory conditions which I have reported above in connection with the treatment of iritis. The structure of the vitreous forbids it. The same may be said of certain forms of scleritis, a disease which, in rare cases, when treated along general lines without reference to a definite cause, leads to disastrous results to the eye. In this connection I have no very striking case to report, but I have seen pleasing modification of a chronic scleritis follow removal of tonsils whose crypts were diseased. The type of choroiditis present in this case occurs relatively frequently; in my experience perhaps not less so than iritis. Until I knew the pathology of the diseased tonsillar crypts and understood from it the analogous conditions existing about the diseased root of a tooth, I was at a loss to find a definite cause for the choroiditis, and to explain the marked tendency to recurrence. If I have drawn clearly the picture of the constant passage into the blood of the micro-organisms and their products which is taking place from the diseased tonsillar crypt; how at times the numbers are few and at other times many more, and how at times the blood conditions are more favorable than at others for combating these invasions, then I have made intelligible the explanation, as I see it, of the tendency to recurrence which exists in these cases.

GLAUCOMA AS A RESULT OF HERPES ZOSTER FRONTALIS, WITH REPORT OF CASES.[1]

By Dr. JOHN E. WEEKS, New York.

AS is well known, herpes zoster, wherever it may appear, is due to a pathologic condition of the branch, branches, trunk, or root-ganglion of the sensory nerve, either cranial or spinal, which supplies the affected area. Since the sensory nerve supply to the eyeball is almost, if not quite, wholly derived from the nasal branch of the supraorbital division of the trigeminal nerve, it follows that we may expect to find a manifestation of the affection of this branch in other areas of its distribution, namely, on the skin of the nose beyond the emergence of the terminal branch, between the lower border of the nasal bone and the cartilage, in all cases in which the eyeball is affected, and observation has proved this to be so in the large majority of cases observed. The sensory nerve supply to the eye is by way of the short ciliary nerves from the ciliary ganglion, whose sensory root springs from the nasal branch of the trigeminal and from the long ciliary nerves also derived from the nasal branch of the trigeminal. The various manifestations of herpes zoster in the eyeball are a keratitis and an anterior uveitis or iridocyclitis, at times mildly involving the entire choroid. The report of the cases follows:

Case I.—J. R., male, aged sixty-two years. At the request of Dr. Colman W. Cutler I saw the patient in consultation on March 20, 1910. Five weeks previously an attack of herpes zoster frontalis began on the left side. The patient had suffered much pain and now presents many deep

[1] Read at meeting of American Ophthalmological Society, 1917.

scars in the skin of the forehead and bridge of nose, extending over part of the area supplied by the nasofrontal branch of the trigeminus. The patient was still suffering some pain throughout the affected area.

The corneal epithelium was roughened over the central portion particularly, and the cornea was hazy throughout this area from infiltration of small cells. Iris congested. Anterior chamber about two-thirds normal depth. Pupils of moderate size, responding very sluggishly and but little to the stimulus of light. Tension + ½. The use of pilocarpin and hot bathing were agreed upon as local measures. Efforts were being made to improve the patient's general condition. March 24, 1910, Dr. Cutler found the tension of the left eye to be still increased.

The condition of the cornea improved very slowly. The tension fluctuated somewhat, but remained generally high. Iridectomy was advised, and on April 20th, at the request of Dr. Cutler, I admitted the patient to the New York Eye and Ear Infirmary for the purpose of operating. V. L. E. = counting fingers at 12 feet. Field of vision slightly contracted (50°) nasally; color-fields considerably contracted. Cornea, central portion hazy; surface slightly roughened. Very slight congestion of ocular conjunctiva. Tension +.

April 21, 1910, iridectomy under ether anæsthesia. A large coloboma was obtained. Healing uneventful. The tension became normal and remained so.

July 31, 1911, tension left eye normal. Vision with the correction of the error of refraction $\frac{20}{70}$. There is still some haziness of the cornea. No cupping of the optic disk. Fundus normal. The right eye does not show any evidence of glaucoma; field normal for form and color. V. with correction = $\frac{20}{20}$.

Case II.—Male, aged forty-nine years, South Orange, N. J. At the request of the patient I met Dr. T. Y. Sutphen in consultation at the patient's residence on December 6, 1915. Dr. Sutphen has kindly permitted me to make a short report of the case here.

A moderately severe attack of herpes zoster frontalis began on the left side about November 12, 1915, confining the patient to the house, the nasofrontal branch being involved. On November 26th the patient began to notice obscuration of vision, which occurred in the morning, but would largely disappear in the afternoon. The obscuration increased from day to day, accompanied by moderate pain referred to the periorbital region. The patient was seen by Dr. Sutphen November 30th. V. L. E. with sph. + 1.25 = $\frac{20}{100}$; R. E. with sph. + 1.25 = $\frac{20}{20}$ +. Patient advised to remain in

bed. Salicylates internally and eserin locally. On December 6th, when seen by me, the cornea was slightly steamy. V. = $\frac{10}{200}$. Fields slightly contracted nasally. Pupil moderately dilated and oval. Tension by tonometer, 40*mm*. Anterior chamber more shallow than in the fellow eye. Disk not cupped. Right eye apparently normal. Tonometer T. 13*mm*.

In view of the steady increase in the glaucomatous symptoms operation was agreed upon and the Lagrange method was recommended. Recently Dr. Sutphen kindly furnished the following notes:

"I made a Lagrange upon that eye December 7, 1915. There was no accident. Tension was at once reduced and remained so. He left the hospital in about ten days. I have mislaid the later notes, but remember that his vision in the left eye was, as he said, as good as ever. Two weeks after leaving the hospital he went south, after telephoning me that he was all right, and I have heard nothing from him for more than a year."

Case III.—W. G., male, aged sixty-five years, came to the New York Eye and Ear Infirmary September 26, 1916, with acute glaucoma in the left eye. On September 20th the forehead and temple on the left side became hyperæmic and painful. Vesicles appeared on September 22d, and the case went on to the development of a typical herpes zoster frontalis, with involvement of the nasal branch of the first division (supraorbital) of the trigeminal. On September 23d the left eye became slightly painful, and on September 26th the patient came to the Infirmary.

Status præsens.—Left eye, lids normal. Ocular conjunctiva congested and slightly thickened. Cornea uniformly hazy. Pupil very slightly dilated. Some minute fibrinous deposits on Descemet's membrane in the lower part of the cornea. Anterior chamber moderately deep. V. = $\frac{20}{100}$. Tension 43*mm*. Diagnosis, acute glaucoma. Treatment, eserin solution, one-third per cent., three times daily.

Right eye, tension 19*mm*. V. = $\frac{20}{30}$ —. No evidence of glaucoma. Patient was ordered a cathartic at night, followed by a saline in the morning. September 28th: Patient admitted to the Infirmary. Tension left eye 47*mm*. V. = $\frac{20}{200}$. As the miotic did not reduce tension and vision became much reduced, a Lagrange operation was performed. Healing progressed favorably.

October 16th: Eye quiet. Tension R. E., 12*mm*; V. = $\frac{20}{30}$ +. Tension L. E., 10*mm*; V. = $\frac{20}{40}$ —. October 29th: R. E., tension 18*mm*; V. = $\frac{20}{30}$ — ; L. E., 13*mm*.;

V. = $\frac{20}{40}$ +. There has been no herpes of the left cornea at any time.

A number of writers mention the fact that increase in tension sometimes accompanies herpes zoster frontalis. Elschnig[1] calls attention to the possibility of the development of glaucoma in herpes zoster frontalis.

Erdmann,[2] in reporting three cases of increased tension (secondary glaucoma) occurring in connection with disciform keratitis, mentions the increase of tension in some cases of herpes zoster ophthalmicus, and endeavors to find an analogue between the affections of the cornea due to disciform keratitis and herpes zoster.

Bubois,[3] in the proceedings of the Holland Ophthalmological Society, December 17, 1911, reported a case of acute glaucoma occurring in a typical case of herpes zoster frontalis. Fearing complications, operation was delayed, but fourteen days later, as the pain and high tension were not relieved by miotics, an iridectomy was performed. The healing was uneventful. V. = 0. A glaucomatous cupping of the disk was present. In the discussion that followed Romunde was of the opinion that because of the presence of a glaucomatous excavation the case was one of simple chronic glaucoma on which an acute attack was grafted. Nicolai mentioned the possibility of bringing on an acute attack in a simple chronic glaucoma by the irritation occasioned by a herpes zoster, and stated that a direct relation between herpes zoster frontalis and glaucoma was not recognized. He also said that iridectomy should have been performed at once.

Dr. Page[4] mentions two cases of increased tension: Case 1, male, aged sixty-four years, herpes zoster frontalis, left. On the tenth day there was mild iridocyclitis with increase of tension. (No mention of invasion of the nasofrontal branch or of the subsequent course of the increase of tension was made.)

Case 2, male, aged seventy-five years. Herpes zoster

[1] Axenfeld, *Diseases of the Eye*, p. 375.
[2] *Zeitschr. f. Augenheilk.*, 1909, p. 30.
[3] *Klin. Monatsbl. f. Augenheilk.*, 1912, p. 601.
[4] *Rec. d' opht.*, 1904, p. 209.

frontalis, right, without involvement of the nasofrontal branch. At the end of three weeks serious iritis right, with slight increase in tension. Aqueous humor cloudy, eye much inflamed and painful. Cupping after scarification and pilocarpin were resorted to. The eye did not recover. No operative procedure is reported.

Dr. F. Verderame[1] reports the following case:

February 12, 1913: Female, aged fifty-four years. Herpes zoster frontalis, left, with involvement of the nasofrontal branch of about four weeks' duration. When seen, the left ocular conjunctiva was slightly congested; some deposits of fibrin on the posterior surface of the cornea. Vitreous body slightly hazy. V. = $\frac{8}{10}$. Tension by tonometer 25–26. R. E. normal tension. Prescribed hot compresses and aspirin. February 22d: Supraorbital pain, left. Injection was marked. Globe a little sensitive. Tension 25*mm*. Deposits on Descemet's membrane were numerous. Pupil circular. Advised continuation of treatment. March 21st: Patient suffers from an exacerbation of the inflammation of the eye. Eye painful. Cornea hazy, but the surface is not stained by fluorescein. Tension 25–26 (right eye, tension 19). April 1st: The condition of the eye is improving. April 5th: Tension left eye 28*mm*. From this time on the condition of the left eye continued to improve until, on December 2, 1913, when the tension was 19*mm* and the interior of the eye approached the normal.

Etiology.—It is now a well-known fact, established by the research work of Dr. Uribe y Troncoso,[2] Stanculeanu,[3] and others that the character of the aqueous humor changes in all cases of congestion or inflammation of the uveal tract from whatever cause, and in some cases of inflammation or injury to cornea and sclera. Troncoso found an increase in albumin and the acquirement of substances preventing the free passage of aqueous into the channels of egress.

Stanculeanu found increase of both organic and inorganic matter in the aqueous in glaucoma, especially of the acute type; also in iritis, parenchymatous keratitis, in other inflamed conditions of the eye, and after some subconjunctival

[1] *Annali de ottal.*, 1913, p. 772.
[2] *Archiv d'opht.*, xxx., pp. 9–151.
[3] *Ophthalmoscope*, vol. xiii., p. 263.

injections. In a case of glaucoma secondary to a tubercular uveitis the writer obtained a decidedly positive test for globulin in the aqueous. Since the cases reported occurred in elderly individuals in whom the anterior chambers were approximately two-thirds of the normal depth, it is probable that the lymph-spaces at the filtration angle were none too large to permit of the filtration to take care of the normal outflow under normal conditions, although there had been no previous increase of tension in either eye of any of the patients. When, in addition to this condition of the lymph-channels, the aqueous has added to it albuminoids, globulin, and possibly colloid substances, rendering it less diffusible or less able to pass through animal membranes, it is apparent that increase of tension could readily follow and persist to an extent rendering operation necessary. It is the writer's opinion that the determination of high intraocular tension in the reported cases was directly due to the changes in the aqueous humor of the nature mentioned above.

The three cases reported were all cases of monocular glaucoma, with no evidence of glaucoma in the fellow-eye. The first case was one of congestive glaucoma of the subacute type, complicated by herpes corneæ; the second case, one of intermittent congestive glaucoma of moderate severity; and the last, one of acute glaucoma. In all, the nasal branch of the fifth nerve was involved. Operation was thought desirable in all the cases because of the continued reduction of vision and of the persistence of other symptoms.

THE TREATMENT OF DETACHMENT OF THE RETINA, WITH SPECIAL REFERENCE TO MÜLLER'S RESECTION OF THE SCLERA.[1]

By Dr. ERVIN TÖROK, New York.

THE treatment of detachment of the retina, with our present procedures, is very unsatisfactory. If we take into consideration the fact that there are cases of detachment recorded where the reattachment took place spontaneously our present methods of treatment seem to be still of less value, since there is none now known which gives better results. With each of the various procedures recommended, be they medicinal or operative, it is only occasionally that we find a good result recorded, most of the cases being failures.

The treatment is most symptomatic, because the ætiology of detachment is still unknown. There are various theories as to the cause, but none of them is universally accepted, indicating that each one has its fallacy. I believe it is unnecessary to enumerate them all here, because they are mostly in oblivion and only of historic value.

The theories that have stood the test of time are Leber's theory, which explains the detachment from a diseased and shrinking vitreous, and the so-called "exudation and transudation theory" of Schweigger, Horstman, and Schmidt-Rimpler, which explains it by a choroidal exudate or transudate which pushes the retina away from the choroid. There is no doubt that both are acceptable to certain cases, and that in some the detachment is due to the shrunken vitreous and in others to something that pushes the retina away from the choroid. In other words, there is no doubt that the cause

[1] Read at meeting of the American Ophthalmological Society, 1917.

of the detachment is not the same in every case, but in various instances there are different underlying causes. Thomson (1), in reviewing the literature on this subject, finds the following mentioned: Myopia, frontal sinusitis, erysipelas, diseases of the uveal tract, heart disease, nephritis, albuminuria in pregnancy, diabetes, lues, rheumatism, gout, exanthemata, malaria, tuberculosis, relapsing fever, pernicious anæmia, trauma, lightning, sympathetic origin. Whatever the cause of detachment in a given case may be, I believe, with Blaskovics, that, from a practical standpoint, we can classify them in two main groups: one where there is a disproportion between the contents of the eyeball and its coats, and another where this disproportion does not exist. In the former group belong those where the cause lies in the shrinking of the vitreous, as detachment following an iridocyclitis, iridochoroiditis, or intraocular hemorrhage and resulting proliferating retinitis, etc., and also those cases where the extension of the sclera, with or without shrinking of the vitreous, is the underlying cause, as in high myopia. In the latter group, where there is no disproportion between contents and coats of the globe, belong those cases where the detachment is due to a choroidal exudate, hemorrhage, tubercle, tumor, transudate, as, for instance, in Bright's disease, arteriosclerosis, or detachment following a contusion of the eye, head, or body.

It is, therefore, very important to study with careful observation and physical examination, the causes in each individual case, as the prognosis as well as treatment depends wholly on the cause. The cases belonging to the latter group are those where cure, either spontaneously or with treatment, could be expected. It is possible that a subretinal transudate, due to arteriosclerosis or Bright's disease, will absorb, if we can beneficially influence the patient's general condition; a traumatic detachment, which, I believe, in cases not due to hemorrhage, differs only in degree from Berlin's œdema of the retina, may get well and the transudate absorb after the circulatory disturbances, due to the paralysis of the vasomotor nerves of the vessels of the retina and choroid, disappear; a detachment due to choroidal exudate may be cured with antiluetic or tuberculin treatment.

Two cases that came under my observation recently may be of interest here:

One is that of a woman, forty-nine years of age, with marked arteriosclerosis and high blood-pressure. She came to me in March, 1915, with a detachment of the retina above and temporally. The eye was soft. She was admitted to the hospital; rest in bed, subconjunctival injections, pilocarpin sweats, and potassium iodide were prescribed. After six weeks in the hospital no change was observed, and she left with a vision of counting fingers at a distance of three to four feet excentrically. Two months later she returned, stating that two days previously her sight suddenly returned. On examination we found no detachment, tension normal, the eye apparently well. V. = $\frac{20}{30}$. This condition remained so for three months, when she suddenly again lost her sight and returned with a soft eye and total detachment. Since then the eye has taken the usual course of this condition, and at present she has posterior synechia, cataract, and no perception of light. Two or three months ago detachment developed in the other eye, too, and patient at present is totally blind.

The other case is that of a young man, thirty years of age, slightly near-sighted, who had lost his right eye from detachment of retina several years ago. The fundus of the eye is not visible, the lens being opaque. He came to us in May, 1915, complaining of failing vision in the left eye. This eye is also slightly near-sighted. In the periphery of the fundus there are many old and recent choroidal patches and several rather circumscribed, small areas where the retina is detached. He was taken to the hospital, where he reacted positively to 3*mgm* of old tuberculin. The reaction was local and general, but no focal reaction could be observed. He was then treated with tuberculin B. E., and under this treatment not only the recent choroidal patches became absorbed, but the many areas of detachment also became reattached. His vision now is $\frac{20}{30}$ with glasses; he has returned to his usual occupation as a clerk, and has been doing his work for over a year.

The second group, where there is a disproportion between the contents and the coats of the globe, is the one where the prognosis is much less favorable. Unfortunately, this is the group where most of our cases belong, since the greater proportion of the detachments are those due to high myopia.

Medicinal treatment, as is well known, is of no avail. There are scores of operative procedures recommended for these cases. Kittel, as early as 1860, recommended the paracentesis of the sclera, and it is still the method most in favor with the majority of ophthalmologists. There are various modifications of this procedure: some puncture only the sclera and choroid; others include the retina. Pagenstecher makes many multiple small stab-wounds; Wolfe makes one large one. De Wecker uses the electrocautery; Parinaud, Higgers, and also Holth, the trephine. This procedure has lately again been recommended by Parker (2) and Thomson and Curtin (3) who perform it without puncturing the choroid or retina and aspirate the subretinal fluid with a hypodermic syringe, at a later date repeating the aspiration as often as necessary. Others, as De Wecker, Galezowsky, Martin, and Evers, endeavor to keep the wound open and to produce constant drainage of the subretinal fluid by introducing gold or silver wire, rubber, horsehair, etc.

Then, again, we find ophthalmologists who try to produce reattachment by producing inflammation between retina and choroid, so as to make the two membranes adhere to each other; such is the electrolysis, injection of solution of iodin or potassium iodide in the subretinal space, or even in the vitreous (Abadie), and also the punctate cauterization of the sclera, as recommended by De Wecker and lately by H. Dor. None of these procedures, however, as experience shows, has given good result, and consequently they have been abandoned. There is really no possibility of getting permanent results with them, since they do not in any way influence the disproportion existing between globe and contents. Deutschmann (4) seems to be the first who realized this principle. He says, in describing his method of cutting the vitreous and retina, that a detachment can become reattached only if the subretinal fluid is removed, but, at the same time, there is enough fluid introduced into the vitreous, *i. e.*, preretinally, to compensate for the removed fluid; therefore he does not remove it, but empties it in the vitreous chamber. Later on, he went still further, and after removing the subretinal fluid, introduced rabbit's and calf's vitreous into the vitreous chamber. In other words, Deutschmann realized

that to cure a myopic detachment we have to endeavor to equalize the disproportion between contents and globe, and he tried to achieve it by increasing the volume of the contents. That the principle was sound is shown by the fact that his results are better than those hitherto obtained with any other method. Elschnig (5) says that up until 1907 Deutschmann had about 400 cases of detachment of the retina under treatment. He succeeded in curing with his operations 24.7 per cent of the cases, while with other methods Uhthoff obtained only 6.1 per cent. Deutschmann, however, had not many followers. His first operative procedure is not practicable, since it has to be repeated several times—sometimes as often as 30 to 40; after each operation the patient must be kept in bed for two to three weeks, and the interval between operations is also several weeks, so that the whole treatment may last for years. The second method of injecting rabbit's or calf's vitreous is often accompanied by very severe inflammations. Although Deutschmann considers these very beneficial, still it is a symptom which is objectionable and keeps many ophthalmologists from performing this operation. At the same time we know at present that the fluid injected into the vitreous cannot become a part of it, especially if it is taken from an animal.

L. Müller (6), in 1903, published his new operation, by which he tries to relieve the existing disproportion by excising a piece of the sclera and thereby diminishing the size of the eyeball. According to his theory the detachment in high myopia is due to the stretching of the choroid with atrophy and circulatory disturbances, followed by a transudate. He says that he came to this conclusion because, while performing his operation, he found a yellowish fluid between choroid and sclera, a fact that I can also confirm. He attributes the beneficial effect of his operation, therefore, not to the diminishing of the size of the eyeball, but to relieving the tension of the choroid, thereby improving its blood circulation and removing the cause for a transudate to form between choroid and retina. The operation, as I have performed it, differs slightly from the original operation of Müller. This modification was advised by Blaskovics (7) and is as follows:

The patient is put under a general anæsthetic, although,

according to Blaskovics, this is not necessary, as the most important and delicate part of the operation, the resection of the sclera, is not felt by the patient. I, however, have performed all these operations under ether. The operation begins with an extensive canthotomy, after which the speculum is inserted and the conjunctiva incised above the external rectus muscle. The conjunctiva is carefully dissected off of the eyeball and then the external rectus is seized. Two catgut sutures are inserted into the muscle, which is then severed between them. An elliptic space of about 20*mm* in length and 10*mm* in width is now outlined with a scalpel on the sclera. The anterior border of this area is behind the insertion of the external rectus and the posterior border toward the equator. The elliptic incision is carried halfway through the thickness of the sclera, and the superficial layers of the flap are removed. Five sutures are now inserted, using double armed threads and suturing from within out. The sutures are raised out of the way, and the posterior border of the incision is carried through the entire thickness of the sclera, taking great care not to injure the choroid. To avoid this, it is better practice to penetrate at one point with the knife, as in cyclodialysis, and afterward to enlarge the incision with the scissors. The choroid is now separated from the sclera, then it is punctured, and by drawing the sutures together, the anterior and posterior wound edges of the sclera are united and, at the same time, the scleral flap is pushed into the pocket between sclera and choroid with the aid of an iris spatula. The sutures in the external rectus are now tied, and the wound of the conjunctiva and skin closed. Atropin is instilled, both eyes bandaged, and the patient put to bed, where he remains on his back for eight days. Both eyes are bandaged for three days, after which the non-operated eye is left open. The operated eye is bandaged for nine to ten days. The sutures of the conjunctiva and skin are removed on the sixth day. The sutures of the sclera, of course, are not removed and they remain there without causing any disturbances. In one of my patients they have been in the sclera for over three years and are well visible under the conjunctiva. In my first two cases I used catgut sutures for the sclera, but found them impracticable, because only the finest catgut

can be used, owing to the small needles employed. This catgut is not strong enough and easily breaks when the sutures are tied. The reinsertion of a suture, after the eyeball has been opened, is extremely difficult and almost impossible without rupturing the choroid and losing some vitreous. The reaction following the operation is very slight. In two or three cases we found a considerable œdema of the bulbar conjunctiva and also of the upper lid. This, however, subsides in a day or two. Müller mentions intraocular hemorrhage as a complication, but I have so far not seen this. The external rectus muscle recovers its function entirely within about two weeks. When the first ophthalmologic examination can be made, about five to six days after the operation, the scleral flap, which is tucked in the suprachoroidal space, is fairly well visible as a well-defined, curved white line, and in some cases the choroid and retina are thrown into several folds at this place.

The operation, although first published in 1903, has not found many followers; there are, at least, very few cases published, Müller (8) reports 10; 6 of them recovered. Holth (9) operated on 3, with 2 good results. Czermak (10) reports 1 unsuccessful case. Schultz (11) reports 1, where reattachment occurred and the vision improved, but later an anterior cortical cataract developed. Blaskovics (7) reports 6 cases; 4 were for myopic detachments: in 3 the results were good and permanent (under observation for one and one-half years); in 1 the detachment recurred six months later. Leitner (12) reports 2: in 1 he had no result; in the other it was satisfactory.

Elschnig (13) presented one cured case to a medical society in Austria, and reported at the same time that he had performed this operation in 11 instances. His results were, however, not given. I performed my first operation in 1912, and since then operated upon 11 cases. In my first 6 I had no permanent results. The detachment in each case was much smaller, or disappeared entirely immediately after the operation, but recurred later. In one of these the patient's sight improved from hand movement to $\frac{20}{50}$, but a month later the detachment recurred and the sight was again entirely lost. I attribute these failures to lack of technic and

to the fact that I excised but a very small piece of the sclera—not larger than 10 by 5*mm*. As I later on increased the size of the excised scleral flap the results were better and more promising. Following are the cases and their short history:

L. M., a woman of forty-five years of age, who lost one eye from detachment of retina due to high myopia, came to the Herman Knapp Memorial Eye Hospital Clinic in July, 1914, with high myopia and detachment of one month's standing in the other eye. V. = fingers at six feet. July 14, 1914, Müller's scleral resection was performed, and to date she has a small shallow detachment below, which interferes but very little with her field of vision and her sight. V. with correcting glasses = $\frac{20}{100}$.

H. G., male, forty-seven years old, high myopia in both eyes. Right eye lost from detachment several years previously. Came under observation in October, 1914, with a detachment of six months' standing in the left eye. The detachment was almost total, and the patient's sight was only movement of the hand in front of the eye. October 27, 1914, Müller's resection. Immediately after operation he had no detachment and vision was $\frac{20}{100}$ with glasses. Later the detachment recurred, and in January, 1915, he had a rather large one in the periphery; field concentrically contracted to about 20 in all directions. V. = $\frac{10}{200}$; no improvement with glasses. This condition is stationary up to this date.

A. F., female, thirty-eight years of age. Right eye high myopia. V. with sph. — 8 = $\frac{20}{30}$. Left eye myopic, detachment of eight months' standing, almost total detachment, including the macula. Vision: hand movements excentrically. February 20, 1917, Müller's resection of the sclera. Immediately after operation no detachment, field free, but has a central scotoma. V. = $\frac{10}{200}$ excentrically. Condition is stationary up to this date. Small circumscribed detachments can be seen in the periphery.

J. H., male, thirty-eight years of age, high myopia. Right eye, detachment eight months. V. = fingers at three feet. The detachment is shallow, but extends over the entire fundus, not including the macula. March 1, 1917, Müller's resection of the sclera. No detachment after operation; field free. V. with glasses = $\frac{20}{100}$. To date, below in the extreme periphery, small, very shallow detachment. V. = $\frac{20}{100}$.

H. S., a male, twenty-eight years of age, high myopia both eyes. Left eye, total detachment for five months. V. = movements of hand excentrically. Scleral trephining performed, but without result. February 6, 1917, resection of sclera. Very large scleral flap excised—about 25 by 15 *mm.* Immediately after operation no detachment of retina. V. at present with sph. — 5 = $\frac{10}{200}$.

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REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

Annual Congress

The Annual Congress of the Society took place in London, on May 3rd to 5th inclusive, chiefly at the home of the Royal Society of Medicine. The Chair was occupied, at all the meetings, by Mr. E. Treacher Collins, F.R.C.S. In addition to the reading and discussion of set papers, a large number of clinical cases were exhibited and debated, and operations at several of the Eye Hospitals of the Metropolis were witnessed by members and visitors. The proceedings also included an inspection of St. Dunstan's Hostel for the Blind, in Regent's Park, and a discussion there on **employment for the blind.** In connection with the Congress an exhibition of macroscopic and other specimens and models of ophthalmoscopes, arranged to illustrate the history of the instrument, was held, in charge of Mr. McMullen, who read a paper dealing with the history of development of the ophthalmoscope.

In his inaugural address, the President referred in sympathetic terms to the demise of the President, Mr. Walter Jessop, Consulting Ophthalmologist to St. Bartholomew's Hospital, during his term of office. His death would, Mr. Collins said, be keenly felt by all ophthalmologists, for he labored hard in his specialty, and latterly did much to establish and make a success the new British journal devoted to it, as well as at the scheme for affiliation of the various Ophthalmological Societies of this country.

If, said the President, in some future generation, an ophthal-

mologist wished to see what part his forerunners played during these important three years of the history of the country, he would find it fairly reflected in the transactions of this Society, as the predominating topics of debate at this, as at the previous two, concerned injuries arising in warfare. The longer the war lasted, the more valuable did the skill of the ophthalmic surgeon become, and it was being increasingly utilized.

He proceeded to discuss **the sequelæ of hypotony,** and gave the following as his conclusions:

(1) That hypotony may occur in connection with several different affections of the eye, and that the sequelæ to which it gives rise may be considered apart from the affection causing it.

(2) That the sequelæ of hypotony vary in different cases according to the chamber—whether the aqueous or the vitreous —in which the pressure is most reduced, and according to its degree, duration, and the rapidity of its onset.

(3) That these sequelæ may be classified as follows:

First, those due to relaxation of tissues usually kept taut: (a) wrinkling of the anterior elastic lamina of the cornea, producing superficial double-contoured lines; (b) wrinkling of the posterior elastic lamina of the cornea and layers of the substantia propria immediately adjacent to it, producing seated striated opacities; (c) thickening of the sclerotic and the production of puckers in it behind the insertions of the recti muscles; (d) wrinkling of the elastic lamina of the choroid and heaping up of the pigment epithelium on its inner surface, where the folds are deep; (e) displacement forwards of the lens and slackening of its suspensory ligament, both of which tend to increase the refraction of the eye.

Second, those due to the production of relatively higher blood-pressure, compared to intraocular pressure, than exists under normal conditions. (a) In the choroidal veins the normal excretion of lymph through them is arrested, and, instead, an exudation of lymph takes place, giving rise to detachment of ciliary body and choroid. (b) In the veins of the iris, the normal excretion of the aqueous humor through them is arrested, and, instead, an exudation of lymph into the anterior chamber takes place. This exudation alters the normal consistency of the aqueous by increasing its percentage

of proteids and rendering it spontaneously coagulable. (c) In the capillary vessels of the ciliary body, the secreting function of that structure is quickened, and an exudation of lymph takes place, the albuminous constituents of which, in a rabbit's eye, being unable to permeate the epithelial lining of the part, raise it up in the form of vesicles. (d) In the capillary vessels of the choroid, the normal exudation of lymph is stimulated, and, if it permeates the pigment epithelium, it may collect beneath the retina and cause its detachment. (e) In the retinal vessels of the optic disk the normal excretion of fluid from the vitreous is arrested, and an exudation of lymph into the head of the optic nerve takes place, causing the appearances of choked disk.

Lieut.-Colonel R. H. ELLIOT said hypotony might exist for months in those who had had sclero-corneal trephining performed, without there being indications of disability. Other conditions besides a direct wound of the lens during operation might determine a cataract. He doubted whether operative procedures ever caused lenticular opacities. The problem of the eye was one of hydrodynamics, not one of hydrostatics, and the manometer was not sufficiently delicate to indicate the minute differences of pressure existing in the eye. He agreed that the tendency towards myopia after trephining was only transitory. In one case he saw, there was as much as 2½ diopters of myopia within a fortnight of the procedure. As the flap consolidated and the chamber deepened, the lens system receded, and the myopia became corrected.

Mr. HERBERT PARSONS thanked the President for having insisted on the importance of minus tension, a subject too little attended to in the literature. He did not agree with Colonel Elliot as to the slight results of hypotony when continued. Though more delayed, they were as bad as those of increased tension.

Mr. W. H. MCMULLEN, who was in charge of the Museum, read a paper on the **history of the development of the ophthalmoscope.**

Captain W. WALLACE examined, in the light of many statistics, the effect on efficiency in the Army of the **altered standard of vision for recruits** which was set up by the War Council, and a resolution on the subject was agreed to.

Captain R. R. CRUISE demonstrated to the Congress his **visor for the protection of the eyes of soldiers,** and contributed a very interesting paper on the subject. He said his hope was to afford to the eye of the soldier a measure of protection comparable to that which the steel helmet had given to the head generally. The War Office having adopted his steel visor, it was now having an extensive trial in warfare. The preventable cases of eye injury included those in which a small shell splinter penetrated the globe, but did not pass through it. In some of the preventable cases, the cornea was incised, the iris prolapsed, and a septic panophthalmitis ensued, the foreign body having had sufficient energy to penetrate the globe, but not to completely traverse it. In the same class were contusions of the globe, dislocating the lens, traumatic cataract, irido-dialysis, intraocular hemorrhage, and retinal lesions. He had seen only a few cases of detachment of retina during the war. Morex and Moreau, of Paris, collected 488 cases of eye injury caused by shell, bomb, or grenade, and estimated that 54% were caused by small particles. They considered that half the cases of eye injury were avoidable by wearing a gauze shield. A large series collected by Captain Cruise himself gave 58% as preventable injuries.

His visor consists of a transparent mesh of steel rings, which have been rendered rust-proof; these are so closely linked together that only very small particles can enter the interstices, and then only if the impact is at right angles. The visor is permanently attached to the steel helmet, and in such a way that it can be easily drawn to either side, or thrown on to the top of the helmet out of the way. The advantage it possesses over goggles is twofold: no obscuring layer of moisture collects when the man perspires, and when struck by a fragment there is no splitting up of the protecting body, as in the case of goggles, and the consequent risk of splinters entering the eye.

After several congratulatory speeches, the Congress agreed to the following resolution:

> "The Ophthalmological Society of the United Kingdom, having examined the steel visor designed by Captain Cruise, has every reason for believing that it would prevent at least 50% of the eye injuries received in warfare. The visor has

received the strong approval of ophthalmic surgeons in the field and at home. The Society begs respectfully to urge that its production should be expedited, and that its application and method of use should be placed under the supervision and control of a competent authority."

Mr. J. GRAY CLEGG discussed the question of **sclerocorneal trephining for hypertension,** and gave the results he had achieved in 250 cases.

Discussion on Employment of the Blind.

Members visited, in the afternoon, the College for the Training of the Blind, which was founded by Sir Arthur Pearson (who is himself sightless) in Regent's Park, and is known as "St. Dunstan's." Here they saw the methods of training adopted in fitting the blinded heroes to earn their own living in the world by means of handicrafts or clerical occupations, while ample provision is made for the recreative and social side.

Sir ARTHUR PEARSON, in initiating the discussion, said St. Dunstan's had set a new pace in the blind world; indeed, he had been interviewed by a deputation from another large institution with similar aims to learn how the training was accomplished in so short a time as seven to eight months. He insisted that these afflicted young men should be encouraged to banish from their minds all morbid reflections as to their condition; that not only increased their happiness, but greatly facilitated the mental concentration needed for re-education. The employment in this work of blind pupil-teachers was of great assistance, owing to the sympathetic viewpoint it gave. Actual study was restricted to five hours daily, in two equal portions before and after the midday meal, reading and recreation—in which boating on the park lake was included,—and everything was done to make the men feel they were active members of the world. Many had passed the searching examination and received the certificate of the Incorporated Society of Masseurs, and were giving very great satisfaction in the work they were doing in various establishments. A sense which was highly cultivated by blind people was that of proximity to obstacles. Sir Arthur regarded it as consisting of three factors; echo, differences of air pressure,

differences of temperature. The first of these was absent in a carpeted room, and the third was only slightly operative, though, in the open, all were important. The abiding danger to a blind person was a door left half-open, and where there were blind people every door should be kept shut when no one was actually passing through the aperture. It was always best to walk erect, then if an upright obstacle were encountered, the foot struck it first.

Mr. ARNOLD LAWSON, who has acted as expert colleague of Sir Arthur throughout this great work, entered into the subject in some detail. He said it had become a work of national importance. The aim of the institution was to secure centralization of effort: previously there had been much overlapping. The young blind man had come to be trained, and he stayed until he had been trained, no time-limit being laid down. The important After-care Department followed up and looked after the blinded man when he went out into the world. Any mistakes he made needed prompt correction, otherwise they became a habit without the worker's knowledge. He needed protection against unscrupulous persons, and help in the buying of materials.

In conclusion, Mr. Lawson dealt with the question of marriage for the blinded man. He advocated this course in cases where a suitable partner could be found, as the blinded man needed, in a peculiar degree, hands to keep him tidy and be an encouragement to him. This hostel, brought into being by the war, would prove a great agency in the future to all who might suffer the affliction.

Mr. HAROLD GRIMSDALE spoke from his extensive experience at the Normal College for the Blind. The craftsman's handicap when deprived of sight was greater than that of the professional man, who depended more upon his brain, the integrity and capacity of which was not reduced, but might be quickened, by his infirmity. His memory was usually improved, and Sir Arthur Pearson himself was a good illustration of that. It was a mistake to concentrate on one kind of employment for all blinded people; variety should be sought, as all were not capable of success in any one form. About 85% of the blinded men who had been at the Normal College had been taught to be self-supporting.

Captain A. W. ORMOND said that in 1911 there were 45,000 blind people in the United Kingdom. He advocated the training of women to run hostels for a few blinded men each; these would be homes for young blinded men. Every effort should be made to render blinded heroes from the war as presentable as surgery was capable of, and the importance of this should be emphasized to the victims of the misfortune, so that they would submit themselves to whatever procedure would accomplish that end.

The PRESIDENT proposed a cordial vote of thanks to Sir Arthur Pearson. He said that when this discussion was proposed, some people asked what the Society had to do with that; it was even hinted that a doctor was better at the bedside than at a funeral. The analogy, however, was not quite apt. When a patient died, there was an end to his sufferings; when a person lost his sight, some of his worst sufferings were only just beginning. The ophthalmic surgeon often had to pass on a patient the sentence of loss of sight, but he had still to treat the patient, or some doctor had, and, if that were possible, to hold out some means of hope in mitigation of that sentence. This visit, and especially seeing how cheerful the people were, enabled them to do that.

Friday was devoted to witnessing operations at several ophthalmic hospitals and discussing a long series of clinical cases. The "Bowman Lecture" was delivered by Sir George Berry, on "Color-Sense Phenomena, and Some Inferences which they Seem to Suggest."

In the evening a number of papers were delivered and discussed.

Concussion Injuries to the Visual Apparatus.

The closing sitting was devoted to this subject, which was opened by Captain A. W. ORMOND, R.A.M.C. He based his thesis on a large number of cases seen by him at one of the large military hospitals and at St. Dunstan's Hostel. He said that the concussion following modern explosives might lead to very definite lesions. Violence communicated through the bones of the skull could affect the inner tunics of the eye. Blows on the sides of the orbit could be transmitted through

the sclerotic, and cause serious concussion of retina or choroid. Air vibrations could influence the part of the eyeball which was unprotected by bone. There had been many cases of serious damage to the globe in which no solid particles appeared to have struck the eye—*i. e.* "windage" cases. Vibrations which had not the force to disintegrate the dense sclerotic might yet produce rupture of the more delicate choroid and retina. There were four ways in which concussion effects could be produced: (1) by vibrations the result of metal or other particles traversing the orbits; (2) by vibrations the result of blows on the bones of the orbit and face transmitted to the interior of the eyes; (3) by vibrations transmitted to the deeper parts of the globe through the sclerocorneal tunic without rupture of the eyeball; (4) by vibrations the result of windage. He gave records of a number of cases in each category to support his submissions.

For treatment, he had great faith in the electric hot-coil; as a reliever of pain he did not think it had an equal. For hastening the absorption of vitreous or other hemorrhage he believed mechanical means were better than any others. He dealt with complete traumatic cataracts as soon as the eye had recovered from the immediate effects of the traumatism. In treating wounds which involved the air cavities flanking the orbits, free drainage was necessary. Frequent hot fomentations were best for wounds in this region, which were mostly septic.

Dr. S. A. Kinnier Wilson dealt with these concussion injuries in the cases in which the trouble was of central origin. The whole subject of commotion was still shrouded in considerable obscurity, and there was some looseness of conception. In some fatal cases, the most minute investigation did not reveal an anatomical lesion. In most cases of a severe blow on the head there were capillary hemorrhages, minute lacerations of the cortical substance, and the consequent disintegration of myelin. Even severe symptoms, however, might be completely recovered from. The experiments of Alan Newton showed there was an organic basis for the transient disturbances of function seen in so-called "railway spine," and allied conditions. A surprising amount of tissue change might arise from even a feeble impact. Cases of pure

commotio were rare, owing to the tremendous speed of modern projectiles. The nearest case of the pure condition he had encountered was that of a soldier, æt. 19, who was struck on the back of the head by a shell fragment while preparing wood for a fire in his dug-out. His three minutes' spell of unconsciousness was terminated by a friend moving an injured arm. He was quite blind for forty-eight hours. On the third day he could see dimly with each eye. There was no dysmetropsia. Two months later, the pupillary reaction and the optic disks were normal; vision $\frac{6}{6}$ in both eyes, Jaeger 14 in both. There was a concentric diminution of both fields for both white and colors, and there was definite visual fatigue. An X-ray examination showed that the outer table was splintered, and the inner one cracked. It certainly was not a case of traumatic hysteria. Except for the visual symptoms, there was no impairment of function of the nervous system.

In the general discussion, the PRESIDENT, Captain CRUISE, Sir GEORGE BERRY, Mr. A. L. WHITEHEAD, Dr. G. MACKAY, Mr. HARRISON BUTLER, and Mr. CRIDLAND took part, and Captain ORMOND replied.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY FOR THE THIRD AND FOURTH QUARTERS, 1916, AND THE FIRST QUARTER, 1917.

By H. KOELLNER, Berlin; W. KRAUSS, Marburg; R. KÜMMELL, Erlangen; W. LOEHLEIN, Greifswald; H. MEYER, Brandenburg; W. NICOLAI, Berlin; H. PAGENSTECHER, Strassburg; K. WESSELY, Würzburg; and M. WOLFRUM, Leipsic, with the Assistance of Drs. ALLING, New Haven; CALDERARO, Rome; CAUSÉ, Mayence; CURRAN, Kansas City; DANIS, Brussels; GILBERT, Munich; GROENHOLM, Helsingfors; v. POPPEN, Petrograd; TREUTLER, Dresden; and VISSER, Amsterdam.

Edited by MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S., New Rochelle, N. Y.

(*Continued from page 395.*)

XV.—VITREOUS AND CHOROID.

63. BROWN, H. H. **Bony tumor of the vitreous chamber springing from the ciliary body.** *Ophthalmology*, October, 1916.

64. RANDOLPH, R. L. and SCHMEISSER, H. C. **A clinical and pathological study of two cases of miliary tubercles of the choroid.** *Transactions American Ophthalmological Society*, 1916.

BROWN (63, **Bony tumor of the vitreous springing from the ciliary body**) enucleated the inflamed and painful eye of a girl 18 years old, who had suffered since childhood from attacks of severe inflammation. A diagnosis of glioma had been repeatedly made and enucleation advised. The specimen showed a bony tumor entirely within the ciliary body. Whereas bone formation is quite often found, usually following panophthalmitis, the author was unable to find another case of such tumor of the ciliary body and regards the case as an example of fœtal inclusion of bone tissue.

ALLING.

The cases reported by RANDOLPH and SCHMEISSER (64, **Miliary tubercles of the choroid**) are of interest because

one was a case of general miliary tuberculosis with acute tuberculous meningitis, and the other a case of general miliary tuberculosis without meningitis. The tubercles were demonstrated during life and verified at autopsy with finding of the tubercle bacillus in the lesion.

ALLING.

XVI.—GLAUCOMA AND INTRAOCULAR TENSION.

65. ANDREW, J. H. **Spontaneous rupture of the cornea and delivery of the lens in a glaucomatous eye.** *Ophthalmic Record*, March, 1917.

66. GIFFORD, H. **Trephining in buphthalmos. A disappointment.** *Ophthalmic Record*, August, 1916.

67. HUIZINGA, J. G. **An improved Elliot operation for glaucoma.** *Ibid.*, August, 1916.

68. PARKER, W. R. **The sclerocorneal trephining operation for glaucoma. A clinical report of forty-five cases.** *Annals of Ophthalmology*, July, 1916.

69. PARKER, W. R. **The relation of choked disk to the tension of the eyeball.** *Journal American Medical Association*, October 7, 1916.

70. STIEREN, E. **Iridotasis in glaucoma.** *Ophthalmic Record*, February, 1917.

71. TRANTAS, A. **A new procedure of sclerectomy for glaucoma.** *Clinique Ophtalmologique*, vii., 9.

ANDREW (65, **Rupture of the cornea and delivery of the lens in a glaucomatous eye**) relates the case of a man whose eye was severely inflamed and had a deep ulcer at the limbus. It was very hard. Two days later he was attacked in the night with a sudden pain and the lens was extruded. A tear was found in the cornea running from the ulcer and the iris and vitreous presented in the wound.

ALLING.

GIFFORD (66, **Trephining in buphthalmos**) has operated upon two cases of this sort and in both the reaction was prolonged. There was no bleb formation and the tension and vision remained as before. Others have been disappointed in a similar manner.

ALLING.

HUIZINGA (67, **Improved Elliot operation**) suggests a flap of conjunctiva made by two vertical incisions from the corneal edge, about 8*mm* in width, leaving both upper and lower ends attached. The band is drawn aside with a strabismus hook

while the conjunctiva is dissected from the cornea and the trephine opening made. The flap has the advantage of covering the opening without stitching.

ALLING.

PARKER (68, **Trephining operation for glaucoma**) obtained good results in 76% of his cases of simple glaucoma; in 45% of the cases of chronic inflammatory glaucoma. In cases where a previous iridectomy had been performed, iritis followed in 22%. When a complete iridectomy was done with the trephining there was iritis in 13%; with partial iridectomy 53%, and when the iris was left intact there was 62% of iritis. He is inclined to favor iridectomy to be followed by trephining if necessary. ALLING.

STIEREN (70, **Iridotasis in glaucoma**) reports six of these operations with highly satisfactory results. The procedure was first suggested by Bothen and consists in drawing out the iris and leaving it incarcerated in the wound under the conjunctival flap. The prompt and lasting results are probably due as much to the fact that the iris is pulled away from the iris angle on the opposite side as to the filtration obtained.

ALLING.

PARKER (69, **Relation of choked disk to the tension of the eyeball**) has conducted experiments upon dogs and monkeys by introducing sponge tents within the cranium. He concludes that choked disks can thus be produced by increasing the intracranial pressure. When such pressure has been produced, the eye which has the least tension is the first to show the effect on the disk. He also concludes that the situation of the artificial tumor has no bearing on the question as to which eye is first affected. ALLING.

A. TRANTAS (71, **A new procedure of sclerectomy for glaucoma**) uses a knife with two prominences at the middle third of each edge. The prominences are perpendicular on the flat plane of the knife and have sharp cutting edges. The instrument looks somewhat like a writing pen. He prepares a conjunctival flap as for an Elliot operation, introduces his knife as for a Lagrange, and pushing the knife straight ahead the cutting edges of the grooved portion of the knife dissect out a piece of the sclera in one sweep.

SCHOENBERG.

XVII.—RETINA.

72. BLAKE, E. M. **The ætiology of retinitis circinata.** *Transactions American Ophthalmological Society*, 1916.

73. FEINGOLD, M. **Fuchs's coloboma with a loop of retinal vein hidden behind a fold of retina.** *Annals of Ophthalmology*, January, 1917.

74. LAFON, CH. **Bilateral, symmetrical preretinal hemorrhages.** *Archives d'ophtalmologie*, XXXV., 3.

75. MOORE, R. FOSTER. **The retinitis of arteriosclerosis and its relation to renal retinitis and to cerebral vascular disease.** *Quarterly Journal of Medicine*, October, 1916, and January, 1917.

76. RING, G. O. **Concerning sudden obstruction to the retinal circulation in a case of cardiorenal disease.** *Ophthalmic Record*, February, 1917.

77. VERHOEFF, F. H. **A new method of treatment of retinal separation.** *Ibid.*, January, 1917.

BLAKE (72, **Ætiology of retinitis circinata**) found the typical appearances of this disease in a patient who had had an extensive hemorrhage in the macular region, five months before. The case adds to the yet insufficient evidence that the condition always follows hemorrhage.

ALLING.

FEINGOLD (73, **Fuchs's coloboma with a loop of retinal vein hidden behind a fold of retina**) describes a case of inferior conus which showed a peculiar folding of the retina below the conus. Apparently there was a staphyloma at the lower edge of the nerve and the retina was drawn backward and underneath itself during the process of development. A vein was thus carried under a retinal fold and appeared as a loop covered by retina.

ALLING.

RING (76, **Sudden obstruction to the retinal circulation in a case of cardiorenal disease**) observed the case of a man with high blood pressure who had suffered from hemiplegia and from attacks of temporary blindness. He became suddenly blind in one eye and the fundus showed œdema of the retina with red spot at the macula. The arteries appeared as white cords but later refilled. The phenomenon is that of so-called embolism of the retinal artery but is probably a thrombotic process.

ALLING.

VERHOEFF (77, **New method of treatment of retinal separa-**

tion) recommends puncture of the sclera followed by rest in bed for a few days. He thinks that the trephine has no advantage for this purpose over simple puncture. He then employs electrolysis through a needle, making numerous punctures through the sclera and retina, thus causing slight inflammatory reaction holding the retina in place. The conjunctiva need not be dissected up. The needle is allowed to remain about five seconds. The current is obtained from six dry cells of 1½ volts each. He has had opportunity to try the method in but one case, but that was highly successful.

ALLING.

CH. LAFON (74, **Bilateral, symmetrical, preretinal hemorrhages**) reports the history of a case of this nature met with in a woman 34 years old. The bilaterality and symmetrical arrangement, the absence of any vascular lesions, and the extreme rarity of such a condition induced the author to report this case.

SCHOENBERG.

To Marcus Gunn the credit is due for having first accurately defined those characters of the retinal vessels which are indicative of retinal arteriosclerosis, and for showing how close is their relation to cerebral vascular disease, but it has been reserved for MOORE (75, **Retinitis of arteriosclerosis**) to point out that there is a form of retinitis which is associated with general arteriosclerosis, caused by the local retinal vascular disease, and only incidentally associated with disease of the kidney. He claims that the retinitis is in large measure distinct from renal retinitis in its ophthalmoscopic characters, in its significance, and in its prognostic value. It is always associated with severe general arteriosclerosis and retinal vascular disease; its gradual evolution from a condition of retinal arteriosclerosis can be traced; the ophthalmoscopic appearances are in a large measure distinctive; it is frequently unilateral and cotton-wool patches never occur; the tenure of life of the patients is uncertain, but they often live a number of years; the cause of death is referable to disease of the vascular system, not to disease of the kidney.

The ophthalmoscopic changes described by Gunn were: (1) Irregularity of lumen of the retinal arteries; (2) tortuosity of the arteries; (3) an exceptionally narrow and bright central

light streak which may show a series of brighter points at intervals; (4) loss of translucency of the arterial walls; (5) obstruction of the blood flow in the veins where they are crossed by arteries; (6) œdema of the retina. These points are amplified, in one or two instances modified, by the present writer. A high degree of arteriosclerosis may occur without any irregularity of the lumen; an irregularity when present indicates severe disease and a high or very high blood pressure. The lumen may narrow gradually, as the vessel is traced along, or it may be reduced more sharply. A constriction may remain unchanged for several years. The pressure in a narrowed vessel may be subnormal even when the general systolic pressure is very high. Defects in the visual field may or may not be present. Tortuosity of the arteries is one of the less valuable signs, although corkscrew arteries in the macular region form, when present, a valuable sign. The unusually bright, slightly irregular central light reflex is one of the most important signs, but too much emphasis must not be placed on bright arteries in the absence of other signs. The phenomena to be seen at arteriovenous crossings are never seen apart from arteriosclerosis, are always present in sclerosis of any considerable degree, and the variations in their degree are so easily made out that they form a reliable means of judging the extent to which the sclerosis has developed. These phenomena consist of: (1) evidence of obstruction to the flow in the veins; (2) displacement of the line of the vein. It seldom happens that a marked degree of distention peripheral to the crossing can be seen, but when a vein crosses above a highly sclerosed artery the actual narrowing of its lumen can be seen, and it occasionally happens that a vein is crossed twice by the same artery within a short distance, and then the trapped portion may be much reduced in size. Normally where an artery crosses a vein the line of the vein is not diverted in the slightest. In the earliest stage of sclerosis of the artery the line of the vein will remain unaltered, but the vessel will be hidden for a narrow margin on each side of the arterial wall, but as the sclerosis increases the vein becomes diverted; it may run alongside the artery for a short distance and then pass under it, or it may change its course so as to pass under the artery at a right angle to the latter's

course. These phenomena at the crossing Moore regards as the most valuable and important single sign of severe arteriosclerosis. He does not look upon œdema of the retina as a sign. The arteries may be generally reduced in size until in extreme cases they are converted into fibrous threads. Retinal hemorrhages of various types are common. Optic atrophy may result either through thrombosis, or the gradual progressive cutting off of the supply of blood.

The evidence adduced in support of the proposition that arteriosclerotic retinitis is a clinical entity would need to be reproduced practically in toto to make the argument clear, so reference must be made to the original paper. The writer first discusses the pathology of renal retinitis, and then the characters of the arteriosclerotic form, emphasizing the ophthalmoscopic changes, which are always superadded to the signs of retinal arteriosclerosis, the arrangement and changes in the dots, the sheathing of the arteries, the frequent unilaterality of the disease, and the histological characters of the exudate. The differential points given are: (1) The sclerosis of the retinal arteries is always a precursor to the development of the exudates, and evidence of this is always well marked; in renal retinitis vascular disease is usually present, but less marked and the evidences of it are apt to be masked by the other manifestations of retinitis. (2) Retinal hemorrhages tend to be smaller and more scattered than in renal retinitis. (3) The retinal exudates are to some extent distinctive in appearance and arrangement. (4) Patches of soft-edged exudate are rare in arteriosclerotic retinitis, and when they occur are probably evidence of beginning renal insufficiency. (5) General retinal œdema does not occur, and retinal detachment is rarely if ever produced. (6) The stellate figure may be present in either case. (7) In forty-five per cent. of the thirty-one patients the retinitis was unilateral. Renal retinitis may be evident in one eye before the other, but it seldom remains unilateral for more than a few days or weeks.

Evidence of the evolution of arteriosclerotic retinitis from retinal arteriosclerosis is drawn from the gradual evolution of the disease in an individual; from a comparison of a group of patients in whom retinal arteriosclerosis was present with a group in whom arteriosclerotic retinitis was present, and

from the length of life and manner of death of the subjects of this affection.

The relation of sclerosis of the retinal arteries to cerebral arteriosclerosis is thus summarized: (1) Of 44 patients suffering from a gross cerebral vascular lesion 31, 70%, exhibited evidence of retinal vascular disease, and in 19 of them, 43%, it was severe in degree. (2) Of 66 patients, 35 of whom had retinal arteriosclerosis alone and 30 had exudates in addition, 27 are known to have died, and the cause of death was determined in 26 as a gross vascular cerebral lesion. (3) Of the patients in whom retinal vascular disease was sufficiently severe to give rise to symptoms, there are 46 in whom there is satisfactory information as to the development or otherwise of gross cerebral lesions, and 21 of these had either suffered from such a lesion or developed one in the course of about three years. FOSTER.

XVIII.—OPTIC NERVE AND VISUAL TRACT.

78. DANTRELLE. **A few cases of papillœdema following war injuries.** *Archives d'ophtalmologie*, XXXV., 3.

79. ELLETT, E. C. **A primary intradural tumor of the optic nerve.** *Journal American Medical Association*, July 8, 1916.

80. MICHEAL, J. C. **Report of a case of tabetic ocular crisis.** *Ibid.*, December 23, 1916.

81. WILDER, W. H. **Optic neuritis from syphilitic leptomeningitis.** *Transactions American Ophthalmological Society*, 1916.

ELLETT (79, **Primary intradural tumor of the optic nerve**) removed the tumor through an incision in the conjunctiva on the other side of the eyeball. The nerve was divided in front of the mass and a large part of the tumor removed, but there was evidence of extension backward to the apex and perhaps into the cranial cavity. For some time after the operation there was œdema of the retina, but this disappeared later, leaving a few white dots, deposits of pigment and contracted vessels. Examination proved the tumor to be a glioma.

ALLING.

MICHEAL (80, **Tabetic ocular crisis**) reports the case of a tabetic with optic atrophy who had attacks of stabbing pain in his left eye accompanied by momentary flashes of light. The conjunctiva was reddened and there was lacrimation.

His eye felt as if it were greatly swollen and that side of the face was numb. Ocular crises have been observed before.

ALLING.

WILDER'S (81, **Optic neuritis from syphilitic leptomeningitis**) first patient had swollen eyelids due to œdema and engorgement of the veins, restricted motility, blurred and swollen disks, large and tortuous retinal veins, and absolute blindness. There were constitutional symptoms and signs of meningitis. Wassermann was positive. Under antisyphilitic treatment the vision partly returned, but the disks were white. The syphilitic exudate probably pressed on the chiasm and oculomotor nerve and interfered with return venous flow through the cavernous sinus. The second case, after having a quadrant defect in the field, suddenly developed complete blindness in the right eye. No lesion of the fundus was discernible. There was girdle anæsthesia about the trunk and a doubtful Babinsky sign. Under mercury there was rapid improvement to nearly normal.

ALLING.

DANTRELLE (78, **A few cases of papillœdema following war injuries**) calls our attention to the possibility of the development of a bilateral papillœdema several weeks or even months after cranial injuries. He relates 3 such interesting cases in which the papillœdema appeared in spite of an extensive exploratory trepanation performed before the ocular condition had developed. The author has seen only 3 papillœdemas in 2800 cranial injuries, but thinks that this condition would be met more frequently if the eyes of these patients would be examined at regular intervals, also weeks or even months after the injury occurred.

The late appearance of a papillœdema is of great diagnostic importance and points to a grave intracranial lesion, which has been overlooked. The prothetic surgery for the occlusion of loss of substance in the bones of the skull should be postponed as long as there is any sign of papillœdema.

SCHOENBERG.

XIX.—ACCIDENTS, INJURIES, FOREIGN BODIES, PARASITES.

82. ALTER, F. W. **Postoperative hemorrhage in a hæmophiliac following enucleation of the eyeball.** *Ophthalmic Record*, October, 1916.

83. BLAAUW, E. E. **A case of contusio bulbi.** *Ophthalmology*, July, 1916.

84. BUTLER, T. HARRISON. **The ring magnet.** *The British Journal of Ophthalmology*, January, 1917.

85. FRENKEL, H. **Paracentral scotoma with diminution of visual acuity due to an injury in the occipital region.** *Archives d'ophtalmologie*, XXXV., 4.

86. GOETZ, R. **Treatment of tetanus following traumatic injuries of the orbits and of the eyes.** *Clinique Ophtalmologique*, vii., 4.

87. LAGRANGE. **A case of bilateral blindness of fifteen months' duration following a contusion of the brain. Recovery after a simulated operation.** *Archives d'ophtalmologie*, XXX., 4.

88. LAPERSONNE, F DE. **War injuries of the eyeball by small fragments.** *Archives d'ophtalmologie*, XXXV., 3.

89. MONBRUN, A. **Alterations of the sympathetic nerve in war injuries of the eye and orbit.** *Ibid.*, XXXV., 3.

90. POOLEY, G. H. **The removal of foreign bodies by means of a giant magnet.** *British Journal of Ophthalmology*, January, 1917.

91. ROCHESTER, A. S. **Spontaneous explosion of Snellen's improved artificial eyes.** *Ophthalmic Record*, December, 1916.

92. VELTER, E. **A clinical study of five cases of hemianopsia due to war injuries.** *Archives d'ophtalmologie*, XXXV., 3.

93. VELTER, E., and PERRIN, R. **Intraocular foreign bodies.** *Ibid.*, 4.

94. WHITING, M. H., and GOULDEN, C. **The technic of the Haab and small magnets in the extraction of intraocular foreign bodies.** *British Journal of Ophthalmology*, January, 1917.

ALTER (82, **Postoperative hemorrhage in a hæmophiliac following enucleation**) enucleated an eye which was beginning to develop panophthalmitis as the result of injury. Bleeding continued for fifteen days, during which time sloughing of the tissues of the face about the eye occurred, leaving a large ulcerated surface. There was a family history of hæmophilia.

ALLING.

BLAAUW (83, **Contusion of the globe**) in an eye which had suffered from a severe contusion, found, when the media had cleared, a fanlike mass of lines in the macular region. Similar striations have been described and attributed by Friedenberg to sclerosis and opacification of the nerve fibers. He explains them by the assumption that they are produced by folding of the hyaloid membrane.

ALLING.

The first number of the new British *Journal of Ophthalmology* contains three articles upon the use of the giant magnet

for the removal of splinters of iron from the interior of the globe. POOLEY and BUTLER (90 and 84, **Removal of foreign bodies with magnets**) working in the industrial towns of Sheffield and Birmingham have occasion to use the giant magnet frequently, while WHITING and GOULDEN (94, **Technic of magnet operations**) have had considerable experience with the Haab instrument both in civil practice and at the Base Hospitals in France.

The conclusions reached by them vary so widely that it is evident that the technic of the large magnet has not yet become stereotyped.

A study of the papers shows that there are several questions which demand an answer: Which is the best giant magnet to use? What is the best route for the splinter to leave the eye? Shall we operate at once, or must we wait until the radiographer has localized the fragment?

Pooley at first followed Haab's technic, but his subsequent experience has led him to adopt a modified method.

Haab assumes that the fragments are melon-shaped and smooth. Pooley finds, and all who have worked in industrial districts will agree with him, that they are irregular, triangular, flakes with sharp angles. Most of the splinters met with in Sheffield are fresh from the fire and nearly aseptic. The fragments we deal with at Birmingham are mostly from high-speed steel and fly from a tool which is hot enough to ensure complete disinfection. In consequence, the eye is not likely to become septic. On this account Pooley says that there is no necessity to operate at once, and prefers to wait for a careful localization.

His present practice is as follows:

(1) If the lens be badly wounded, remove the lens, and then extract the fragment by the anterior route.

(2) If the lens have escaped, or be only slightly wounded, remove the foreign body through a scleral incision.

He holds that if the foreign body be drawn forward by the Haab method: (1) the vitreous is liable to become clouded, and pain is caused; (2) the ciliary body is liable to contusion, the capsule is damaged and may be scratched, the iris is bruised, and the inflammatory exudate which covers the foreign body and which may be septic is wiped onto the

damaged lens. This causes the eye to become irritable, and imperils the transparency of the lens; (3) damaged tissues are further injured and their resistance lowered. The advantages of the scleral route are:

(1) The operation is easier.

(2) The inflammatory exudate may come out with the splinter.

(3) The ciliary segment is not bruised.

(4) A sutured scleral wound covered with conjunctiva is less liable to become septic.

The disadvantages of the scleral route are: impaction of the choroid retina and vitreous in the wound: and, though Pooley does not mention it, subsequent detachment of the retina!

Pooley claims that a larger percentage of eyes are saved, that the final acuity is better, and that convalescence is shorter. Pooley's views are open to considerable criticism. We make a point of removing the fragment at the earliest possible moment; we have found the results of localization are so very uncertain, that we never wait if we are certain that the eye contains a fragment. The splinters come out clean, and are not covered with inflammatory exudate, which is the result of waiting for a day or two. This too is the practice of Whiting and Goulden (*vide infra*). Pooley states that the splinters are generally aseptic, as they are, and yet he has much to say about septic exudate.

As regards damage to the lens: Is there any record of damage to the lens by the Haab technic? Colonel Lister, who has had great experience in France and in London, tells me that he has never heard of such an accident.

We gather from Pooley's paper that he adopts the scleral route in every case where the lens is not wounded seriously.

Whiting and Goulden take an exactly opposite view. Working in France at a large Base Hospital they have in one month removed thirty foreign bodies from the globe with the Haab magnet. Their paper is an elaborate description of the technic which they employ to extract magnetic splinters from the eye with the Haab giant magnet used in conjunction with the hand magnet.

If there be a large wound in the sclera they extract by this wound. In these cases the fragment is large, and the eye is

generally so severely injured that there is little hope of saving it. In all other cases they disregard the scleral wound and adopt the original Haab method. The fragment is drawn forward into the anterior chamber, and then removed by a corneal incision with the aid of the small Snell magnet.

The authors have found that in civil practice the results of the Haab route are better than those obtained with the scleral route. In France, where many of the fragments are non-magnetic, to adopt the scleral route after a localization, would entail making an opening into many eyes unnecessarily.

They find that localization is not necessary to successful treatment, and they say that if it involve more than a few hours' delay it is better omitted.

The most modern Haab magnet made at Zurich is the instrument used, and the medium shape-pointed short terminal is always chosen.

Curry and Paxton's Nernst operating lamp has been found to be a great help in the operation.

The position of the patient, and the method by which he is held by the operator, is shown by four photographs.

The difficulties encountered have been the following:

(1) Delay in the passage through the vitreous. This is experienced in cases in which there has been delay, and when, in consequence, there has been time for the fragment to become fixed by exudate. It will be noted that this is the condition which Pooley is likely to meet with, and from his remarks has met with. The methods adopted in such cases are fully described. The magnet is applied to the sclera. The current is broken, and remade; the efforts are continued for as long as five minutes. Time must be allowed to allow the magnet to cool.

(2) Entanglement in the iris. Such a case requires the greatest care in manipulation. The pull must be applied in the plane of the iris, and even through the sclera behind the plane of the iris.

(3) The presence of a small corneal wound with prolapse of the iris. No attempt must be made to use this wound to remove the fragment. It must be drawn to a spot as near the lower angle of the chamber as possible and removed by corneal section.

(4) In the case of a large corneal wound which indicates a bulky splinter the giant magnet must be employed with the greatest caution, and then the fragment removed by the original wound. Any prolapse must be excised after the removal of the foreign body and not before. The authors do not give reasons for this advice which seems open to discussion.

TECHNIC OF THE SMALL MAGNET

The fragment having been brought into the anterior chamber, the patient is removed to the operating table, and generally placed under an anæsthetic. Only when the eye is quite white is this omitted.

The corneal section is now made above the cornea, commencing at a spot half-way between the limbus and the center. The size of the incision will be regulated by that of the foreign body. The terminal of the small magnet is now placed upon the cornea just over the splinter and stroked upward to the wound. It draws the splinter with it and leads it into the wound, whence it is extracted with the point of the magnet. The eye must not be touched with the fixation forceps or the aqueous will escape. If it escape, it may be replaced with saline and the maneuver repeated. It may, however, be necessary to introduce the tip of the magnet into the eye.

THE RING MAGNET

The internal pole magnet designed by Mellinger of Basle is described by the reviewer who has used one for the last four years. An improved balanced model has recently been installed at the Birmingham Eye Hospital, where it has given great satisfaction, and is considered to be a far more efficient instrument than the Haab which it has replaced. Several examples are now being installed in London, and the instrument bids fair to entirely supplant the Haab pattern.

The instrument consists of a solenoidal oval ring just large enough to take the head of a patient. The reviewer finds that it is just a shade too small and might with great advantage be slightly enlarged. The ring is wound with over a mile of insulated wire, and is provided with a rheostat which is not

really necessary though convenient. The ring is balanced with weights, and fixed in a frame in such a manner that it can be lowered over the head of a recumbent patient. The current flowing round the solenoid induces a magnetic field which is homogeneous, and very intense at the center of the ring. Any iron body brought within the oval is strongly magnetized. Soft iron rods are brought towards the eye and eventually placed close to the cornea. Larger and larger rods are employed, till eventually the fragment appears in the anterior chamber. Instead of the eye being brought to the magnet the magnet is brought to the eye. When the splinter is in the anterior chamber, the current is broken, and the anterior chamber opened as described in the former paper except that we make the incision nearer the limbus. A spatule is now employed exactly as the small magnet is used in the Haab operation. The procedure is much simpler than the old method: the patient lies quietly on a pillow, and if necessary is anæsthetized, and the rods are used like any other ophthalmic instrument. The small spatula can be introduced within the eye if the circumstances demand it, and a very much greater power is at command than that furnished by any type of small magnet, such as the Snell or Hirschberg.

The reviewer has never found it necessary to give an anæsthetic, but were it called for he would under no circumstances use chloroform, an anæsthetic which he has for many years ceased to employ. Ether correctly given is perfectly safe and in no way hinders the operator.

The advantages of the ring magnet are fully set out in the paper; they are so great that the reviewer feels certain that none who has ever become accustomed to Mellinger's magnet would ever wish to go back to the Haab.

Measurements of the magnetic flux, and of the actual pull of the magnet would seem to show that the Haab is a little more powerful, but the innen-pol magnet possesses all the strength which is necessary to saturate the fragment with magnetism.

T. HARRISON BUTLER.

ROCHESTER (91. **Spontaneous explosion of Snellen's improved artificial eyes**) has found eighteen instances of such an accident. It usually occurs at the time of extremes of temperature either hot or cold. There is a partial vacuum within

the eye and the glass is under considerable pressure. The fact that the accident has occurred more than once in the same individual goes to prove that in some cases the secretion of the lacrimal sac has a deleterious effect on the glass.

ALLING.

E. VELTER (92, **A clinical study of five cases of hemianopsia due to war injuries**). To the eighty-six cases of various types of traumatic hemianopsia described in the French literature, Velter adds five more interesting histories.

1. Penetrating injury of the left occipital region, right homonymous hemianopsia. Macular vision preserved, hemianopsia permanent.

2. Injury of the right occipital region; bilateral homonymous hemianopsia with macular vision alone left, for some time. Later on, recovery of the right side of the field and finally the patient remained with a left-sided inferior quadrant anopsia.

3. Injury of the occipital region, right side; double bilateral hemianopsia, which changes after a few days into an inferior hemianopsia and later on becomes a permanent right inferior quadrant hemiachromatopsia. It seems that there was primarily a large interhemispheric hæmatoma, pressing upon both areas in the region of the calcarine fissure and producing an extensive disturbance of the field of vision. Later on, after resorption took place, all that was left permanently injured was a small area over the left superior margin of the calcarine fissure.

4. Injury of the left occipital region, right homonymous hemianopsia; gradual recovery of the field, which finally, after three months, became normal.

5. Tangential traumatism of the occiput in the median line; temporary complete blindness for one hour; rapid recovery, with the field normal for white, contracted for colors. Small absolute scotoma of the macula in the upper right quadrant. Pierre Marie and Chatelin have reported three similar cases. The lesion must have been very limited, possibly a puncture by a spicule of bone in the region of the extreme pole of the occipital lobe. These four cases seem to prove the presence of a cortical center for macular vision.

SCHOENBERG.

LAGRANGE (87, **A case of bilateral blindness, of fifteen months' duration, following a contusion of the brain. Recovery after a simulated operation**) relates in detail the case of a soldier who after having seen a comrade struck by a bomb became unconscious for about half an hour and on waking pretended to have lost his sight entirely. The examination revealed nothing abnormal (neurologically and ophthalmologically). The condition had lasted for fifteen months, and Lagrange, who was asked by the military authorities to pass an opinion on this patient's condition, made up his mind that it was a case of simulation. He proposed to the patient to guarantee him a cure by a very serious operation on the brain, with the alternative, if he refused the operation, of reporting him to the authorities as a malingerer. The patient agreed to submit to the operation, which consisted of washing the eyes under a general chloroform anæsthesia. The patient began to see light at the first dressing and gradually had to admit that he is recovering his vision in the course of a few weeks following the operation.

SCHOENBERG.

Numerous cases of various types of hemianopsia due to cortical lesions have been reported by French ophthalmologists, but very few cases are on record showing a lesion of the cortex localized in the center of macular vision. One such case of **paracentral scotoma with diminution of visual acuity to an injury in the occipital region** is reported in detail by H. FRENKEL (85). The patient gives the usual history of having been hit by a fragment of a shrapnel in the occipital region, loss of consciousness, temporary blindness. Taken prisoner, he was returned to France several months later. The examination of his eyes revealed V. right $\frac{1}{10}$, left $\frac{1}{10}$; field of vision: bilateral inferior paracentral scotoma. The author believes that these two findings justify those who believe in the existence of a well defined zone in the occipital lobe, where the macular function is to be localized. He thinks that since the condition remained stationary for about thirteen months, there is no probability of any improvement, and figures that the capacity of work of this patient will be impaired to the amount of 90 %, taking as a guide the table of invalidation of the French War Department. SCHOENBERG.

E. VELTER and R. PERRIN (93, **Intraocular foreign bodies**) report two cases with intraocular foreign bodies visible with the ophthalmoscope.

In both cases there was no inflammatory reaction and the vision in one of them remained good. It was not thought advisable to attempt the removal of the foreign bodies.

SCHOENBERG.

In his experience with 1000 war injuries of the eyes LAPERSONNE (88, **War injuries of the eyeball by small fragments**) has seen 20 per cent. with penetrating wounds. Of these, 6 per cent. lost the eye from iridocyclitis.

Half of the number of cases, blind in both eyes has had small foreign bodies in both eyes with multiple penetrating wounds of the eyeballs ending in bilateral iridocyclitis. He did not meet with one single case of sympathetic ophthalmia. The rarity of this occurrence in the present war is due, according to the author, to the aseptic methods of the present times and to the early enucleation, when indications exist. As a preventive measure against the penetrating injuries produced by small fragments, he advises the use of specially devised protective goggles.

SCHOENBERG.

MONBRUN (89, **Alterations of the sympathetic nerve in war injuries of the eye and orbit**) has seen six wounded soldiers who had one eye partially resected with a stump containing a remnant of the posterior pole of the eye. Several months after the operation, when there was no indication of any inflammation in the orbit, the patients began to complain of continuous burning pains on the same side of the face or head, becoming unbearable at the slightest movement; the mentality was somewhat disturbed. At the same time vasomotor and secretory disturbances on the side of the head corresponding to the enucleated eye were also present (constant vasodilatation and marked hypersecretion of sudorific glands).

The author thinks that this condition, which he calls the sympathetic syndrome, is due to an inflammation of the sympathetic fibers entering the ciliary ganglion and of the ciliary nerves, a true sympathetic neuritis. He tried to excise the stump and the optic nerve attached to it in the depth of the orbit, and also the ophthalmic artery containing in its sheaths

the supposed inflamed branches of the sympathetic. But his attempts did not succeed in relieving his patients of pain.

SCHOENBERG.

R. GOETZ (86, **Treatment of tetanus following traumatic injuries of the orbit and of the eyes**) knows of fifty cases of tetanus that originated from orbito-ocular injuries and advises the preventive injections of serum and thorough cleansing of wounds, especially of those soiled by earth or excrements of horses. The points of entrance for the bacillus of Nicolaïer are the cornea, sclera and limbus, orbit, brows, lids, and, very rarely, the conjunctiva.

SCHOENBERG.